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TREATMENT OF HAND INFECTIONS

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An increasing number of patients are hospitalized at our institution with significant infections of the hand. Some of these infections are quite virulent and destructive in spite of, or as a result of, antibiotic therapy.

The severe handicap of a hand which is limited in motion and function is most keenly appreciated by those who have experienced such malfunction and the surgeons who see and treat these patients. Each infection of the hand must be considered as a potential major problem, even though it may appear trivial upon initial examination.

Sensation, grasp and pinch are the three basic functions of the hand (fig. 1). In addition, the hand is capable of a myriad of complex motions and actions, afforded by smooth acting joints, with pliable capsules and collateral ligaments, gliding tendons and precisely balanced action between the long flexors, the long extensors and the intrinsic muscles of the fingers and thumb. These diverse motions permit independence in the care of bodily needs, enable earning power and afford to some the delicate movements of the artisan.

Infection in this complex organ cannot be thought of in the same terms as a localized, subcutaneous abscess of the thigh. In the forearm and hand there is much in a very small area. Infection here may lead to severe limitation of useful function because of the associated pain, stiffness, swelling, joint and ligament contraction and tendon immobility.

Thorough knowledge of the prevention, early recognition, diagnosis and accurate therapy of these infections are required to maintain or regain as near normal function as possible. This is our aim.

We must be constantly aware of the large number of disabling hand infections, which by history, result from minimal injury or develop without known cause. Tiny cracks and fissures are commonly associated with the thickened skin and calluses on the volar surface of the working man's hand. The repeated contacts and trauma to which the hand is exposed each day may deposit bacteria into these cracks and fissures or a tiny puncture wound. A small abscess may form and spread beneath the thickened epidermis. A tiny puncture wound, just 2 to 4 mm. in depth, in a strategic area (*e.g.*, the volar finger creases) may be the source of a fulminating infection.

The acute infections in most of our patients were associated with either minimal trauma or were of unknown cause.

The behavior of hand infections is influenced by the bacterial virulence, the local action of the infecting organism upon the host tissue, host resistance and the local or specific anatomical features of the involved area.

The basic studies of Kanavel,[4] his critical analyses and progressive methods of treatment of hand infections, provided for others a rich background and understanding necessary for the intelligent and proper care of these potentially disastrous infections. Drs. Sumner Koch and

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Michael Mason,[5,7] collegues of Kanavel until his death in 1938, have contributed additional vital information. The experience of Bunnell[2] and Lampe and Netter[6] has also rendered a more clear understanding of these problems.

Their authoritative, vivid, yet often simple drawings and illustrations have afforded the writer a much clearer understanding of this complex organ, both in its normal state and when associated with infection.

Many illustrations appearing in this publication are those of Mason[7] and Lampe and Netter[6] or personal modifications of their basic concepts and visual aids. These illustrations are properly identified in the accompanying legends.*

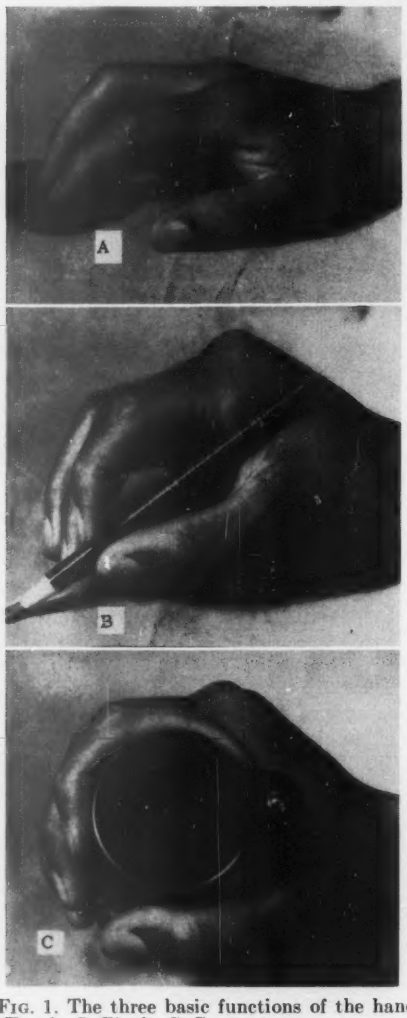

FIG. 1. The three basic functions of the hand. *A*. Touch. *B*. Pinch. *C*. Grasp.

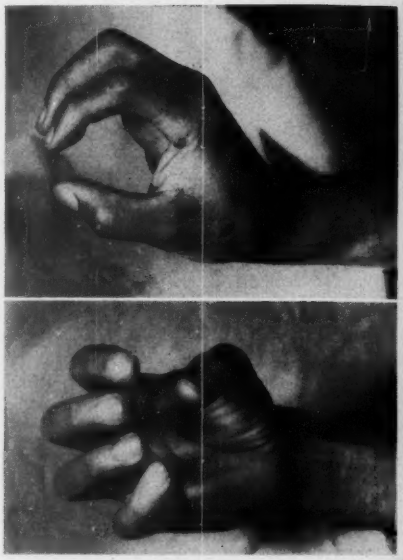

FIG. 2. Position of function. The desired position of the wrist, thumb, and fingers during necessary periods of immobilization.

GENERAL CONSIDERATIONS IN THE CARE OF HAND INFECTIONS

There can be no better treatment for these disastrous conditions than prevention. Safety precautions, proper working conditions, and even good hygiene are important. Prompt reporting of "trivial" accidents, proper examination and treatment by a competent physician will minimize or avoid preventable, serious deformity.

The trivial infection is closely observed.

* The author is indebted to Dr. Michael Mason through Dr. Sumner Koch and Mr. Robert B. Rowan, Managing Editor of the W. B. Saunders Company, who have kindly permitted the use of Dr. Mason's enlightening illustrations appearing in his excellent treatise "Surgery of the Hand" in *Christopher's Textbook of Surgery*, sixth edition, published by W. B. Saunders Company, 1956. The vivid Lampe-Netter illustrations appearing in this article with kind permission of Mrs. L. A. Oppenheim, Ciba Pharmaceutical Products, Inc., are taken from "Surgical Anatomy of the Hand with Special Reference to Infections and Trauma," by Ernest W. Lampe, M.D. and Frank H. Netter, M.D., published in *Ciba Clinical Symposium*, December 1951.

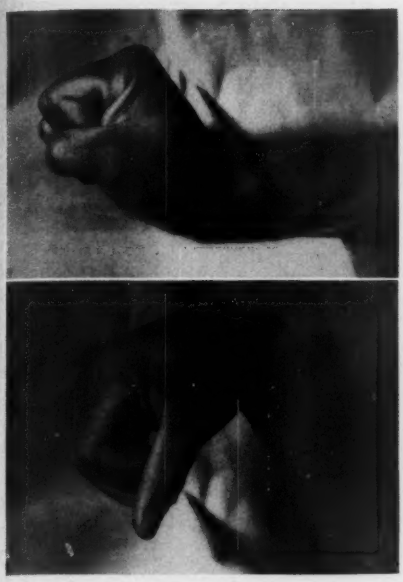

FIG. 3. Position of wrist. Slight extension permits tight fist. Acute flexion prevents tight fist.

During the early phase of inflammation or the spreading phase, conservative therapy is mandatory. Injudicious surgery at this time may propagate the process and, in some cases, may prove fatal. Antibiotics are generously given.

The involved parts, usually the entire hand, wrist and forearm, are immobilized in the position of function (figs. 2 and 3). This important concept, stressed by Kanavel and others, reminds us of the fact that most necessary movements and actions of the hand and fingers are performed from this basic position. The most important digit, the thumb, performs best in the "opposed position."

The metacarpophalangeal (MP) and interphalangeal (IP) finger joints are gently flexed. The MP collateral ligaments are moderately loose when the joint is in the position of partial flexion (fig. 4).

When the fingers and thumb are maintained in the position of function, even minimal motion permits the essential sensitive pinch and grasp: useful function. The swelling, stiffness, pain and immobilization associated with infection often result in fibrosis, contracture, severe limitation of motion and poor hand function. The thumb

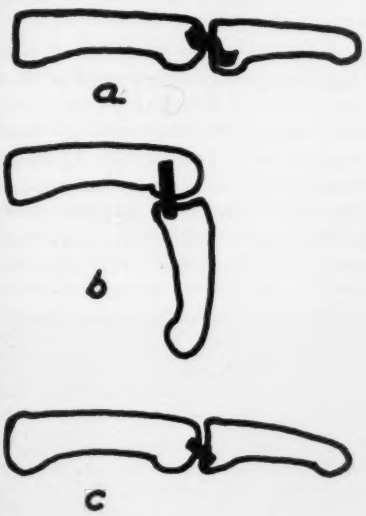

FIG. 4. Collateral ligaments of the metacarpophalangeal joints: *a*. loose and relaxed when the proximal phalanx is extended; *b*. taut when the phalanx is flexed; *c*. contracted, shortened ligaments resulting from immobilization in extension, preventing flexion.

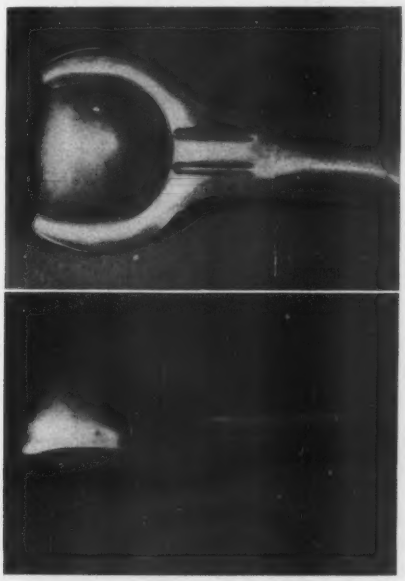

FIG. 5. Universal splint. Permits immobilization of either hand in position of function.

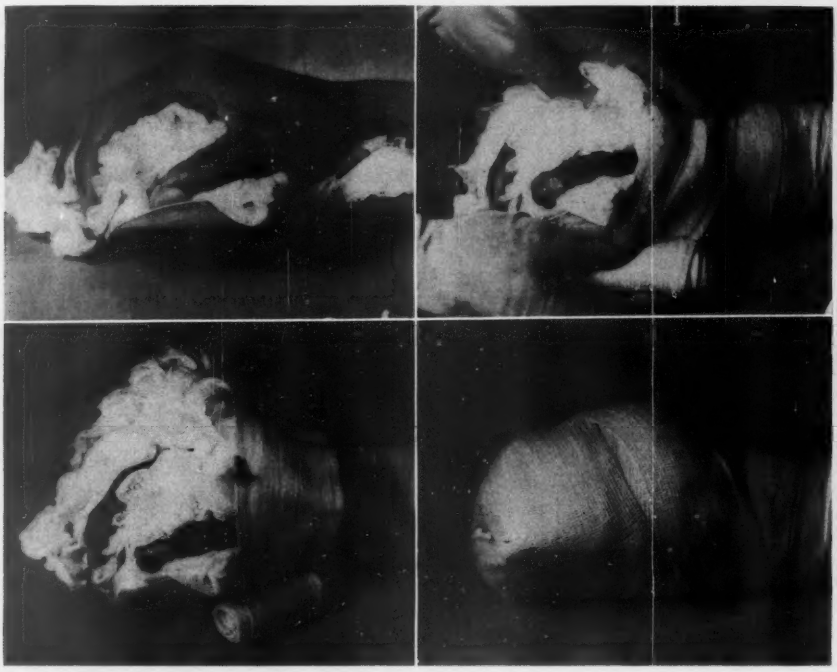

FIG. 6. Immobilization of hand in position of function with universal splint and bulky dressing

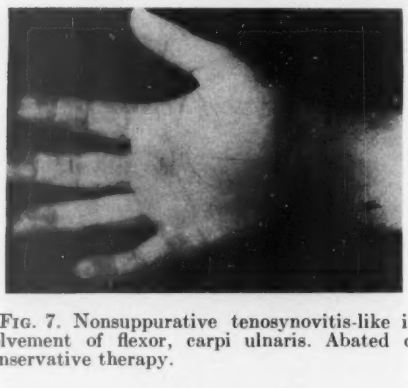

FIG. 7. Nonsuppurative tenosynovitis-like involvement of flexor, carpi ulnaris. Abated on conservative therapy.

must not be carelessly placed in the flat, adducted position, nor should the finger joints (MP and IP) be kept in the extended position for protracted periods. Tightly contracted and shortened MP and IP collateral ligaments rigidly freeze these joints in useless extension. The contracted, narrowed thumb cleft, the fixed adducted, flat thumb are preventable tragedies!

The universal splint effects such useful position of function, when properly used (fig. 5). It is light in weight and permits x-ray studies of the bones without removal of the splint. A bulky, semicompression dressing effectively molds the hand and fingers in the desired position (fig. 6). Moist heat is frequently used.

Conservative treatment, consisting of immobilization, heat and antibiotics, is utilized during the early, cellulitis stage in an attempt to abort the infection and prevent necrosis and pus formation. Frequent observations must be made to determine advance or repression of the infection (case N. S., fig. 7).

Should this regime afford resolution of the process, judicious and rapid return to the full range of active and passive motion is desired. This is accomplished with supervision and proper instruction, and with the patient's full realization that "the rest is up to him." Frequent, almost constant, exercises may be required.

When the infection is localized and an abscess forms, prompt diagnosis and surgical intervention are necessary. This is major surgery, usually employing general anesthesia. The blood pressure

cuff tourniquet provides a bloodless field (fig. 8). Anatomical incisions are used (fig. 9).

Incision and drainage may suffice. In many of our cases, however, adequate incision and exploration have revealed a substantially larger area of subcutaneous and deeper tissues to be involved in a necrotizing process. Judicious, radical debridement was carried out at the time of initial surgery, with careful removal of all overtly necrotic skin, subcutaneous and fascial tissue. When the viability of more vital structures, such as tendons, was at all in question, more conservatism was exercised. If frankly necrotic, however, even these structures were debrided during the first operative procedure. No drains were left in these debrided wounds. The forearm and hand were immobilized in the position of function with a bulky compression dressing and splint.

The first dressing change was performed in the operating room 5 to 7 days later. The clean, granulating wound was secondarily closed or covered with a split skin graft at this time. Again the hand was immobilized in the position of function until the grafted wound was adequately healed.

If additional necrosis was observed during the first dressing in the operating room, secondary debridement was carried out. The resultant clean wound was closed, most often within the next week. Secondary debridement was required on three occasions.

When early closure is effected, a basic tenet is accomplished: "the conversion of an open, infected wound to a clean, closed wound as quickly as possible." The avoidance of chronic drainage, prolonged edema, inflammation and immobilization helps prevent the stiffness, fibrosis, adherence and contractures which are so disastrous to the mobile, precisely functioning hand.

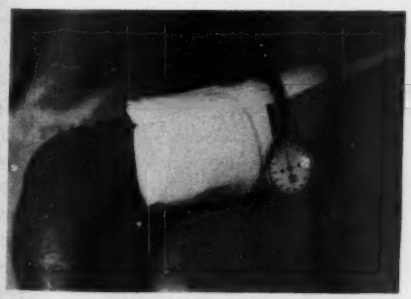

FIG. 8. Tourniquet. Allows safer surgery in bloodless field. Broad blood pressure cuff used with due care.

FIG. 9. Functional anatomical incisions. *A*. Finger synovial sheath, bursae and midpalmar space, Parona's forearm space. "Avoid important motor branch of median nerve." (in *dotted circle*). *B*. Midlateral incision, at tips of flexion creases. "Avoids neurovascular bundle." *C*. Approach to thenar space, Parona's space and radial bursa.

Upon complete wound healing, functional restoration is our goal, with the use of physical,

rehabilitative therapy and dynamic splinting. Should the return to normal function be incomplete after all modalities of treatment, an acceptably functioning hand will result from careful splinting in the position of function. Even less than 50 per cent return of normal range of motion will permit the accomplishment of the three basic functions, *e.g.*, sensation, gross grasp and the all important pinch between the thumb, index and middle fingers.

ANATOMY AND INFECTIONS OF THE FINGERS

Dorsum. The skin over the dorsum of the finger is loose and rather mobile; underlying infection reveals itself by the expected amount of swelling and redness (fig. 10).

Paronychia (fig. 11 *A* and *B*) is an infection under the cuticle at the base or wall of the fingernail. Prompt elevation of the cuticle to release the drop of underlying pus will suffice. Later, once pus accumulates beneath the bed of the nail, surgical elevation of the cuticle and removal of the "floating portion" of the nail are required.

A frequent site of dorsal subcutaneous abscess is over the proximal phalanx (fig. 10). This infection often starts as a follicular infection in this hair-bearing area. The subcutaneous area of pus and necrotic involvement is significantly larger than initially suspected from superficial examination. Such widespread subcutaneous necrosis is shown in case S. I. (figs. 12 and 13).

In case C. U., a severe infection of the thumb developed after minor injury to the dorsal skin

FIG. 10. Common sites of finger abscesses. Volar skin snugly bound to deep fascia by numerous fascicles permits accurate grasp of objects. Dorsal skin and subcutaneous tissue much more loose. *A*. Hair bearing area dorsal aspect proximal phalanx. This abscess begins as folliculitis. The knuckle area is prime location for human bite or human tooth injuries. *B*. Early paronychia. *C*. Advanced paronychia with pus beneath nail. *D*. Subungual abscess usually associated with foreign body such as splinters. *E*, *F*, *G*. Fat pad or pulp abscesses. Advanced forms may involve synovial tendon sheath, may cause osteomyelitis (felon) or may spread dorsally or proximally into deep palmar fascial space via lumbrical canal. Note close proximity of skin at volar creases to the tendon sheath (after Bunnell).

FIG. 11*A*. In its early stage, paronychia starts as a tender swelling in the nail wall which may usually be unroofed easily with a sharp scalpel or scissors without employing an anesthetic. (From Mason, M. L.: *Christopher's Textbook of Surgery.*)

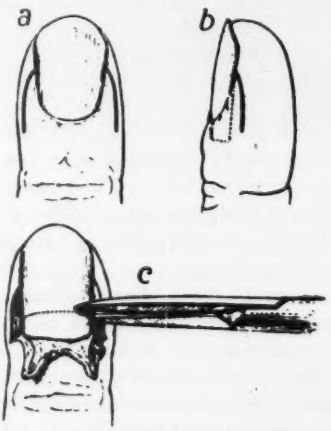

FIG. 11*B*. When the paronychia has extended beneath the nail, it is necessary to raise up the eponychium and excise the overlying nail to secure adequate draining. (From Mason, M.L.: *Christophers' Textbook of Surgery.*)

over the IP joint 1 week before admission (fig. 14, *1*). At the time of prompt surgery, again the degree of deep involvement was much more severe than the superficial appearance indicated. Joint destruction was apparent on physical and x-ray examination (fig. 14, *Op-a, b*). Extensive subcutaneous necrosis and purulent dissection were present on both radial and ulnar sides. The extensor mechanism, joint ligaments and capsule were also destroyed, leaving a flail joint. The

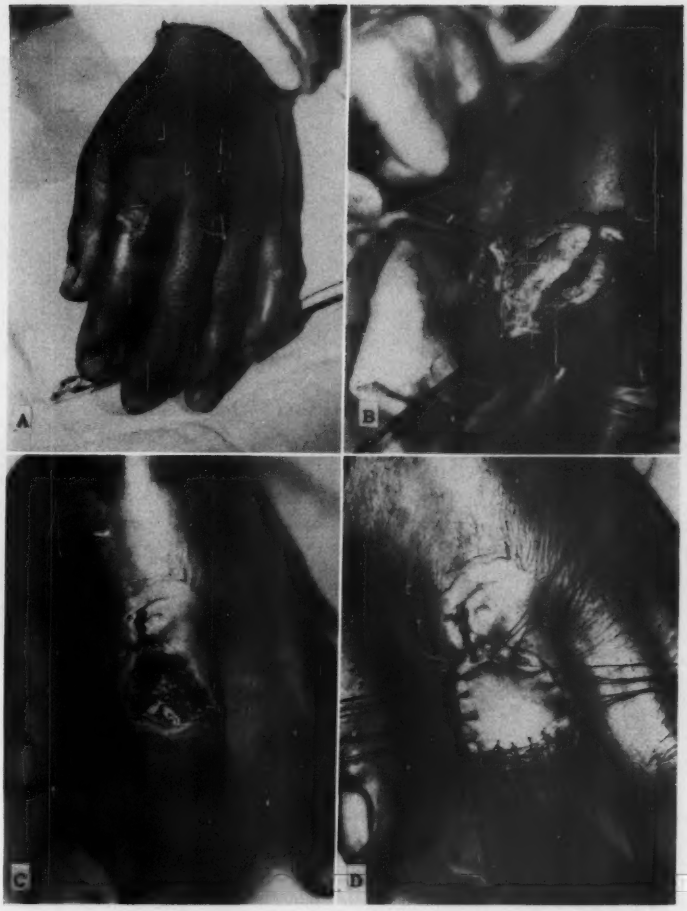

FIG. 12. Dorsal subcutaneous abscess, index finger. *A*. These infections usually arise in the dorsal hair follicles; no trauma. The amount of deeper necrosis not fully appreciated from superficial inspection. *B*. Area debrided. Dorsal hood exposed but not involved in necrotic process. *C*. Appearance of wound 6 days later at second operation. No residual necrosis. *D*. Split thickness graft applied at this time.

FIG. 13. Postoperative healing. *FU*, normal range of motion; 6 months

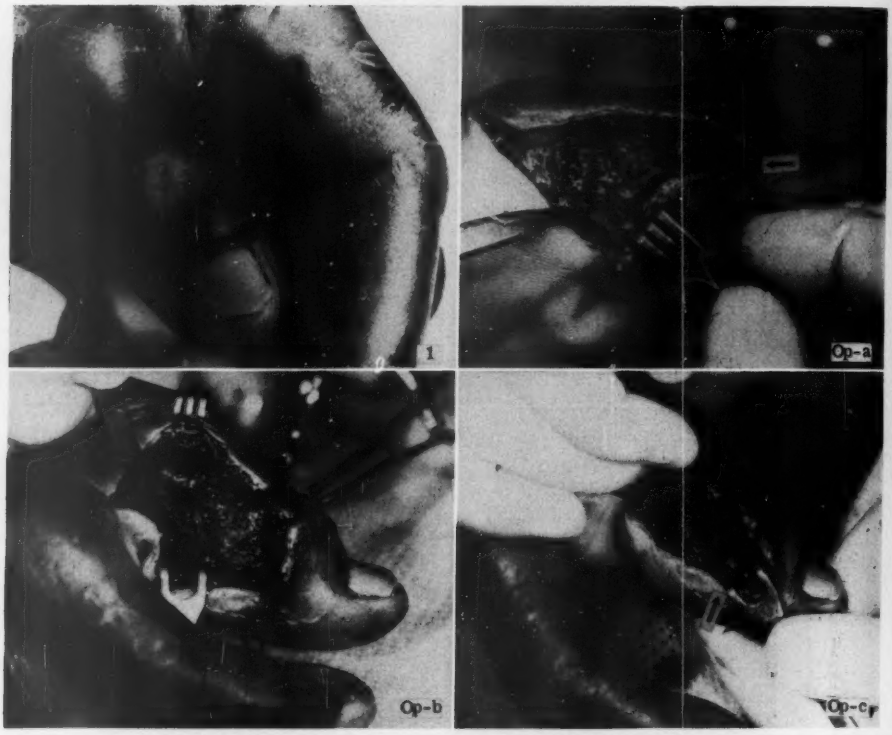

FIG. 14. Infection, left thumb. Much more extensive than might be suspected. Surgical exploration revealed widespread necrosis of dorsoradial (*Op-a*) and dorso-ulnar (*Op-b*) tissues and destroyed IP joint. *Arrow:* joint defect (*Op-a* and *Op-b*). Thorough debridement was accomplished, including joint cartilage and necrotic bone (*Op-c*). *Arrow* indicates cancellous bone ends exposed.

thenar space and flexor tendon sheath were not involved. All overt necrotic tissue was removed at this time, including the remaining cartilage. Cancellous bone ends are indicated by the arrow in figure 14, *Op-c*. The hand was immobilized in the position of function, with due care taken to maintain the thumb cleft, the thumb in the opposed position, and the terminal phalanx in extension.

Prompt healing followed. The interphalangeal joint became ankylosed in the extended position by firm, painless, fibrous union. An excellent functional result was obtained within 2 months (fig. 15).

Human bite or human tooth injuries are most frequently incurred on the dorsum, at the knuckle, the clenched fist striking the opponent's tooth (fig. 16). These infections are vicious and rapid because of the introduction of "human acclimated" bacteria of mixed aerobic and anaerobic type. The perforated tissues are crushed by the blow, and the scant blood supply affords fertile soil for severe infection. The bacteria are disseminated merely by opening or extending the fingers. These wounds should never be closed primarily. The wound is thoroughly cleansed, debrided of all questionable tissue and left open. Divided tendon or joint capsule is not repaired primarily. Definitive local repair is accomplished subsequently, when it is safe.

The angry appearance of a human bite or tooth wound may be observed in case A. B. (fig. 17).

Volar. The surgical attachment of the skin to underlying fascia by fibrous fascicles renders a resilient, nonsliding, sensitive skin over the volar aspect of the fingers (fig. 10). This enables a predictable grasp and pinch and the ability to hold onto objects. These are the most purposeful functions of the fingers.

The terminal vessels and nerves of the finger

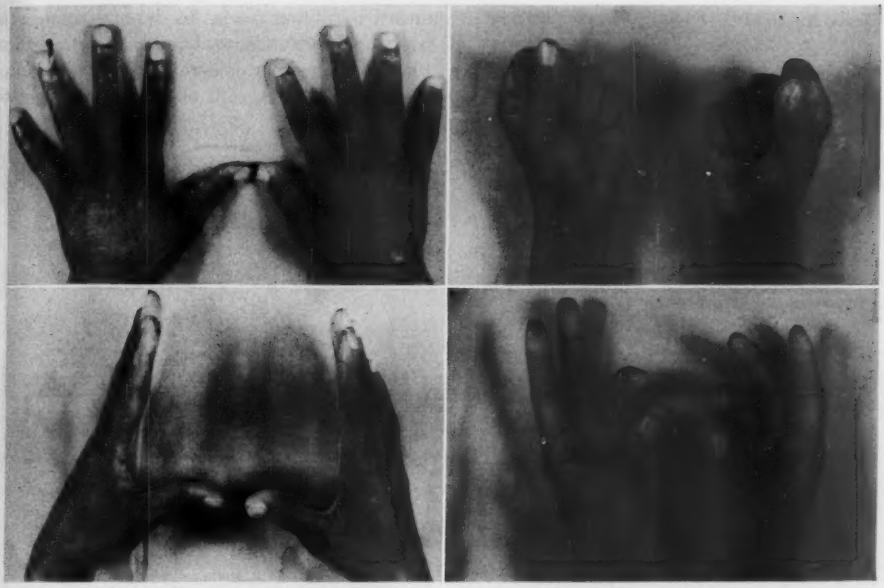

FIG. 15. Follow-up on range of motion, 2 months postoperatively

FIG. 16. Human bite injuries (more correctly human tooth injuries) of the hand usually result from tooth perforation over a knuckle. The tooth penetrates several different spaces in its passage—first, the subcutaneous space, then the subfascial space beneath the tendon, then the joint space. Serious involvement of all these areas soon occurs. (Redrawn from Mason and Koch: Surg. Gynec. & Obst., Vol. 51 From Mason, M.L.: *Christopher's Textbook of Surgery.*)

tip pulp transverse the compartments created by the fibrous septa (fig. 18). These fascial compartments are unyielding, and therefore, the contained infection produces extreme pain but little outward swelling. With time, pressure occludes the terminal vessels with resultant ischemia, infection and terminal phalangeal osteomyelitis—bone felon.

Pulp space infection is usually limited to that space, unless an enlarging process involves the nearby flexor tendon sheath (figs. 10 and 18). Treatment demands early incision and severence of all the septa (fig. 19).

A near catastrophe occurred in the case of S. H., a surgical nurse who pricked the pulp space of her right thumb with a hypodermic needle while administering medication to a postoperative patient with infection in his wound (fig. 20). Signs of local and systemic infection rapidly developed because of the "human strain" of infecting organism. Central necrosis occurred (fig. 20*A*). Radical incision and transection of the septa was performed. In addition, the central volar area of necrosis was debrided (fig. 20*B*). Several days later, a second debridement was required. Fortunately, her recovery was then prompt and she regained full, painless sensation and motion (fig. 20*C*, *D*).

The importance of a normally functioning thumb cannot be overestimated. It is our most important digit. A vigorous and seemingly radical approach at times is indicated to salvage the all important thumb (cases S. H., C. U., B. R., figs. 14, 20, 40).

Extensive destruction may occur in spite of all therapeutic measures, with total tissue or functional loss, as in case K. I. (fig. 21). With widespread damage in a ring or little finger, restoration of useful function may be impossible or not worth the morbidity and the investment of lost time from work. Prompt amputation of the fourth or fifth finger would then be preferable, as soon as this decision is accurately made.

FIG. 17. Human bite. These infections are virulent. "Leave wound open!"

The thumb, index and middle fingers are never amputated, except as a last resort.

The most vulnerable site in the finger for puncture wound contamination to occur is the flexion crease. The undersurface of the skin here is very closely attached to the flexor tendon sheath (fig. 10). These disastrous infections are discussed in detail later.

The majority of the lymphatics of the volar aspect of fingers and palm, except for a small central portion, drain or spill over to the dorsum where more major lymph channels originate (fig. 22). The skin and subcutaneous tissue of the dorsum of the hand are loosely attached to the deep dorsal fascia, in contrast to the volar skin which is snugly attached to the resistive palmar fascia (fig. 10). These two features account for the marked dorsal edema occurring within deep volar infections of fingers and palm (fig. 36).

The acute spreading infections, so vividly described and studied by Koch, have not been seen by us (fig. 23). These dreaded infections, streptococcal in origin, spread wildly via the lymphatics. They are often lethal. Should the patient survive for 5 to 10 days, multiple necrotic areas may develop.

Palmar anatomy and infections. The palmar skin, as in the fingers, is firmly and securely attached by fibrous fascias to the underlying protective palmar fascia, enabling better grasp (figs. 10, 25). The palmar epidermis of the hand is frequently in contact with objects and subject to

FIG. 18. The pulp of the finger is a firm pad made up of interwoven fibrous trabeculae which run from the undersurface of the skin to the surface of the bone. It forms an admirably firm cushion useful in grasping. Since it is unyielding, an infection leads to tense, painful swelling and early blockage of blood supply to the distal phalanx beyond the epiphysial line. (From Mason, M. L.: *Christopher's Textbook of Surgery.*)

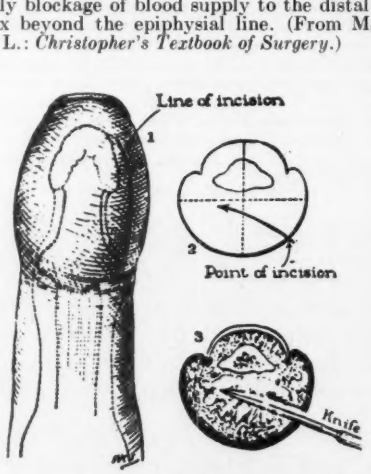

FIG. 19. Drainage of a felon is accomplished by a single hockey-shaped incision, along the side of the phalanx, which is designed to divide the fascial septa. Care must be taken not to make the proximal end of the incision too far proximally lest the tendon sheath be opened. (Koch: J. A. M. A., Vol. 2. Mason, M. L.: *Christopher's Textbook of Surgery.*)

trauma. Infections develop within the thickened epidermis or the thin layer of adjacent fatty tissue. A localized abscess was present in case G. R. (fig. 24).

FIG. 20. Pulp space infections. *A*. Thumb of surgical nurse who accidentally pricked center of pad with hypodermic needle during administration of antibiotic to patient with postoperative wound infection. *B*. Virulent, rapid infection with necrosis resulted (human strain). Two radical incisions and debridements required within 4 days. *C*. Fortunate outcome, 1 year follow-up.

The thenar and hypothenar areas are covered by their muscle fascias and are isolated by the fascial attachments from the central palmar fascia. The tough palmar aponeurosis covers and protects the vital structures which course the central and distal palm (figs. 25, 26). It extends from the palmaris tendinous insertion at the wrist to the proximal interphalangeal joints.

Three groups of volar spaces or channels are anatomically located within or connected with this central palmar cavity. These real or potential spaces are: the deep fascial spaces, the lumbrical spaces or channels, and the tendon sheaths or bursae.

Deep fascial spaces. Fibrous septa extend from the undersurface of the palmar fascia to the deep transverse intermetacarpal ligament and create channels or spaces in which course the neurovascular bundles, lumbrical muscles and the thumb and finger flexor tendons (figs. 20, 28). One key, tough septum extends obliquely, attaching to the third metacarpal, and divides the deep palm (beneath all nerves, vessels, tendons and lumbricales) into two deep fascial spaces: the thenar or adductor space and the middle palmar space (first group, volar spaces).

Lumbrical spaces. The lumbrical muscles, encased in their muscle fascia, provide a second group of potential volar spaces in which infection and pus may accumulate and channel (figs. 26 to 28). The first lumbrical muscle of the index profunda tendon overlies the adductor or thenar deep fascial space (fig. 28). The lumbricals of the mid, ring and little finger tendons abut the middle

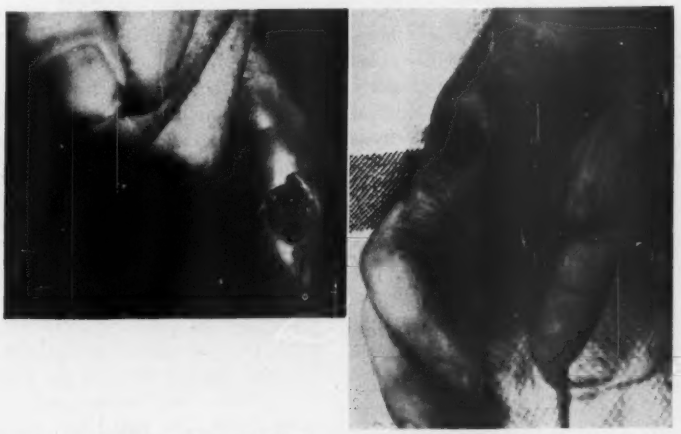

FIG. 21. Severe progressive infection of fifth finger. Repeated surgical debridement; finally amputation. Indications for amputation rare. "Never the thumb."

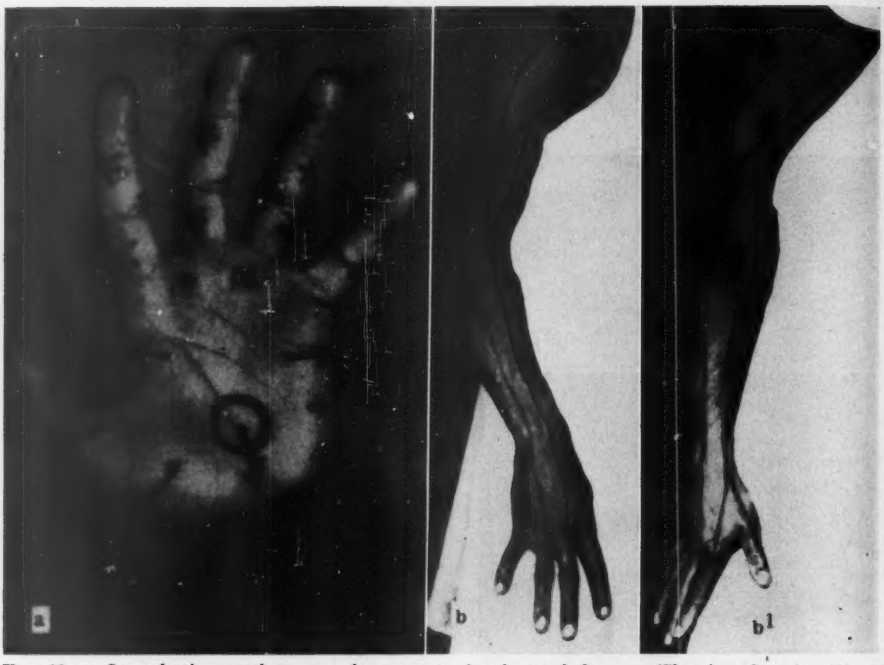

FIG. 22. *a*. Lymphatics, pathways, volar aspect of palm and fingers. (Showing dorsal spillover). *b*. Pathways of lymphatic drainage, dorsum of hand.

palmar space. The lumbrical tendons, *en route* to their insertion into the lateral bands, provide channels through their respective web spaces. Pus may thus extend along the lumbrical canal from its origin in the web space to infect the deep fascial space, or pus in the deep fascial space may point, via the lumbrical canal, into the web space (fig. 29).

Tiny cracks and fissures are commonly associated with the thickened calluses on the volar surface of the working man's hand, particularly at the distal palmar fold. Repeated minimal irritation may introduce bacteria beneath these areas and result in a superficial abscess. As the abscess enlarges, it extends deep to the palmar fascia via its web defects (fig. 25), or it perforates the fascia and forms a large deep abscess (fig. 29). The larger, deeper, purulent component of this collar button abscess may erode through the dorsal skin of the web; it may channel along the lumbrical tendon or canal and infect the adjacent deep palmar space, or infect the adjacent tendon sheath, as in case J. B. (figs. 36 to 39).

Synovial tendon sheaths and bursae. An important third group of volar spaces are those contained within the synovial tendon sheaths of the flexor tendons of thumb and fingers (figs. 26 to 28). These sheaths or bursae provide an excellent gliding surface for the tendons as they move beneath the fascial pulleys at the wrist (transverse carpal ligament) and in the fingers (annular ligaments or fascial sheaths) (fig. 27).

The tendon sheaths of the second, third, and fourth fingers are limited, accompanying the flexor tendons only from the level of the distal palmar crease to the profunda insertion. The proximal portion of the index synovial sheath overlies the deep thenar or adductor fascial space; those of the third and fourth synovial sheaths are adjacent to the deep middle palmar fascial space. The tendon sheath of the flexor pollicis longus, known as radial bursa, accompanies the tendon from its phalangeal insertion up into the wrist, proximal to the transverse carpal ligament (fig. 27). That of the fifth finger extends also to the same forearm level. This same sheath of the little finger encases all eight finger tendons at the level of the proximal palm to distal forearm and is known as the ulnar bursa. The radial and ulnar bursae often intercommunicate at the wrist level (fig. 31). In

FIG. 23. "Acute spreading infection" (Koch) showing "skip areas." Initially the process is virulent, strep, cellulitis with rapid lymphatic spread. "No pus early." "Surgery" at this time may cause death. *A*. Tenosynovitis index finger skip to midpalmar space (not thenar space). *B*. Lymphatic skip spread from dorsum index finger, dorsum hand, forearm, axillary and subpectoral nodes. All areas later became necrotic.

the distal forearm there is a potential space, the retroflexor or Parona's space, which lies between the flexor tendons, their bursae and the pronator quadratus muscle (fig. 30).

The intimate anatomical association and overlap of the three groups of volar spaces are demonstrated in the composite diagram (fig. 31, 35).

Infection in the tendon sheaths or bursae, purulent tenosynovitis, spreads rapidly and destructively throughout the entire individual

FIG. 24. Simple subcutaneous abscess, spontaneous in origin.

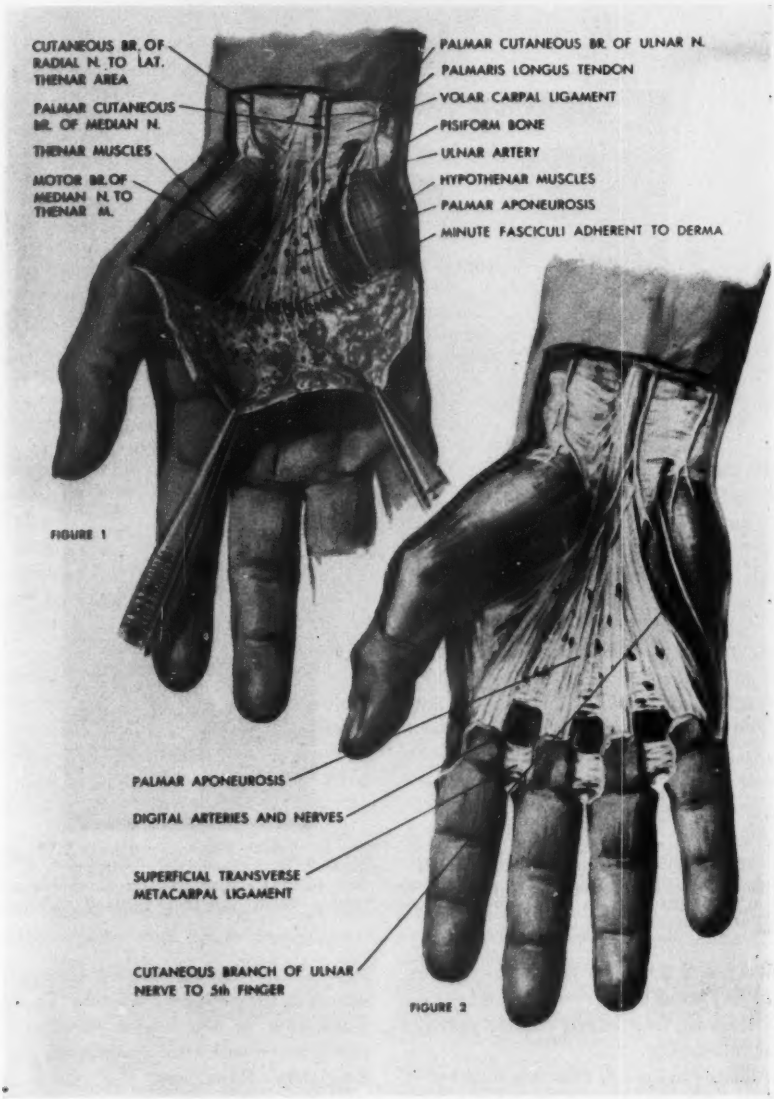
CUTANEOUS BR. OF RADIAL N. TO LAT. THENAR AREA
PALMAR CUTANEOUS BR. OF MEDIAN N.
THENAR MUSCLES
MOTOR BR. OF MEDIAN N. TO THENAR M.
PALMAR CUTANEOUS BR. OF ULNAR N.
PALMARIS LONGUS TENDON
VOLAR CARPAL LIGAMENT
PISIFORM BONE
ULNAR ARTERY
HYPOTHENAR MUSCLES
PALMAR APONEUROSIS
MINUTE FASCICULI ADHERENT TO DERMA
FIGURE 1
PALMAR APONEUROSIS
DIGITAL ARTERIES AND NERVES
SUPERFICIAL TRANSVERSE METACARPAL LIGAMENT
CUTANEOUS BRANCH OF ULNAR NERVE TO 5th FINGER
FIGURE 2

FIG. 25. (From Lampe and Netter: *Ciba Clinical Synposium.*)

RADIAL A. AND VENAE COMITES
RADIAL BURSA
ABDUCTOR POLLICIS BREVIS
TRANSVERSE CARPAL LIG.
FLEXOR POLLICIS BREVIS
SHEATH OF FLEXOR POLLICIS LONGUS
PROBE IN FIRST LUMBRICAL SHEATH
PALMAR APONEUROSIS TURNED DOWN
FIGURE 4
ULNAR ARTERY AND VENAE COMITES
ULNAR BURSA
VOLAR CARPAL LIGAMENT
PISIFORM BONE
PALMARIS LONGUS TENDON
ULNAR ARTERY AND NERVE
DEEP DIVISION ULNAR NERVE AND ARTERY
SUPERFICIAL DIVISION ULNAR NERVE (SENSORY TO 4th AND 5th FINGERS)
MEDIAN NERVE (DIGITAL SENSORY BRANCH)
ULNAR BURSA
SUPERFICIAL VOLAR ARTERIAL AND VENOUS ARCHES
2nd, 3rd AND 4th LUMBRICAL MUSCLES
SEPTA
TENDON SHEATHS
BRANCH OF MEDIAN N. TO THENAR MUSCLES
ABDUCTOR DIGIT V
FLEXOR DIGIT V
OPPONENS DIGIT V
5th FINGER SHEATH
PROBE IN MIDPALMAR SPACE OR BURSA
PROBE IN THENAR SPACE OR BURSA
PROBE IN DORSAL EXTENSION OF THENAR SPACE BEHIND ABDUCTOR POLLICIS
CRUCIATE AND ANNULAR LIGAMENTS
INSERTION OF FLEXOR DIG. SUBLIMIS
INSERTION OF FLEXOR DIG. PROFUNDUS
FIGURE 5
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FIG. 26. (From Lampe and Netter: *Ciba Clinical Symposium.*)

sheath. The increased pressure balloons the sheath and may occlude the scant blood of the tendon, with resultant necrosis (figs. 32, 33). Upon examination, characteristic signs are present, Kanavel's four cardinal signs (fig. 34). Pus within the sheath of the third or fourth finger will rupture into the midpalmar space; from the index sheath, into the thenar space (fig. 35).

The radial and ulnar bursae, containing pus under pressure, serve as conduits for the rapid spread of the infection from the distal phalanx of the thumb or fifth finger to the distal forearm space. Pus in one bursa may infect the other (run-around abscess) (fig. 35); and if under enough pressure, the pus may rupture into the retro-flexor or Parona's space in the forearm (figs 30, 35 and case B. R., fig. 40).

One readily realizes now the manner in which

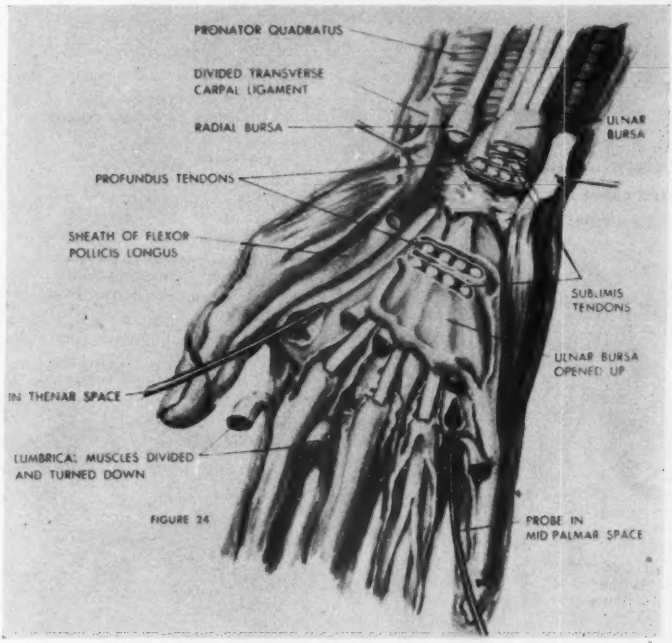

FIG. 27. (From Lampe and Netter: *Ciba Clinical Symposium*)

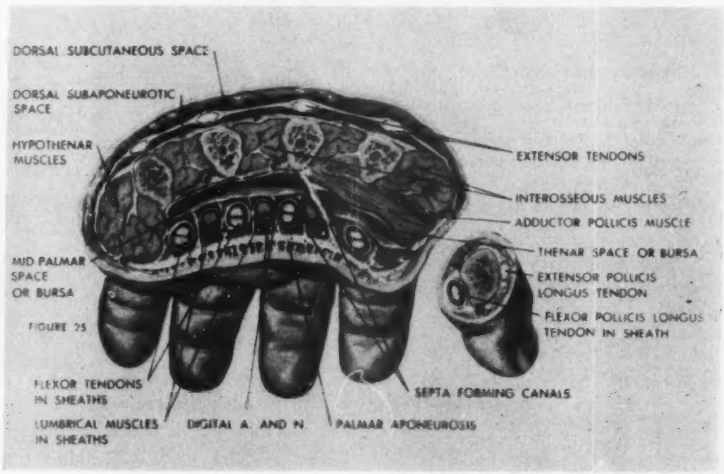

FIG. 28. (From Lampe and Netter: *Ciba Clinical Symposium*)

an innocent appearing, superficial abscess may spread to involve the entire hand and forearm with disastrous results: a functionless, useless hand.

A patient (J. B., fig. 36) presented himself with a deep, palmar abscess, secondary to an infected callus on his left distal palmar fold.

In view of the widespread infection, and especially the cellulitis of the distal forearm, surgery was delayed for 24 hours. During this time mas-

FIG. 29. Collar button abscess. *MC*, metacarpal; *MPS.*, middle palmar space; *LUMB*, lumbricle muscle; *P.F.*, palmar fascia.

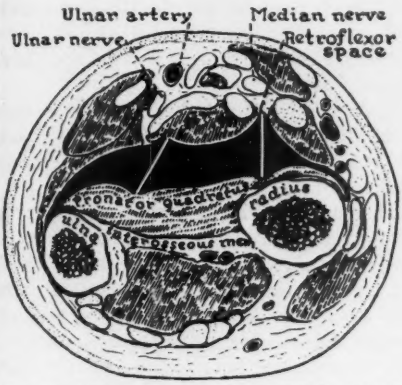

FIG. 30. The relationship of the radial and ulnar bursae to the retroflexor space in the lower forearm. (After Kanavel. Mason, M. L.: *Christopher's Textbook of Surgery.*)

sive amounts of antibiotics were given; the arm and hand were kept immobilized and warm; moist packs were applied.

Surgical exploration revealed gross involvement of the distal palm (fig. 37): both deep fascial spaces, the lumbrical channels, their three web spaces and the synovial tendon sheaths of the mid, ring and little fingers. Incision and drainage of sheaths and spaces, together with thorough debridement of all overt necrotic tissue were accomplished. The flexor tendons were viable.

One week later examination in the operating room revealed clean, granulating wounds; these were promptly closed with split skin grafts (fig. 38). Healing was complete within 1 month (fig. 39). A good functional result was apparent at this time. Though limited somewhat in range of motion, the hand was quite adequate to provide useful function (fig. 39). The factors influencing this result are: temporary 24-hour delay of surgery to permit coverage and treatment with antibiotics, evacuation of pus along with the radical and adequate excision of the necrotic tissue, prompt closure of the wounds and immobilization in the important position of function until healing is achieved.

Case B. R., a bricklayer, represents the most widespread, acute infection in this group of cases (fig. 40). It began in the distal pulp space of the thumb without history of significant injury. Gradually during the next 3 weeks, the process

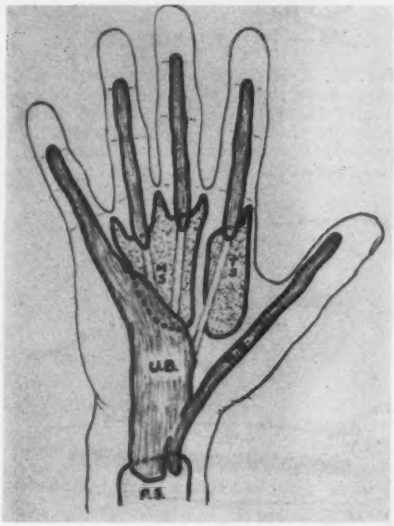

FIG. 31. Composite diagram of the proximity and overlap of the groups of volar spaces. Deep fascial spaces: *M.S.*, middle palmar space, *T.S.*, thenar space. Synovial tendon sheaths of the index, mid and ring fingers ending at distal palmar crease. *R. B.*, radial bursa. Thumb synovial sheath extending proximally into wrist. *U.B.*, ulnar bursa. Sheath of little finger encasing the eight flexor tendons of the fingers in the proximal palm and wrist. Radial bursa and ulnar bursa often communicate with one another and are adjacent to the deep forearm or Parona's space.

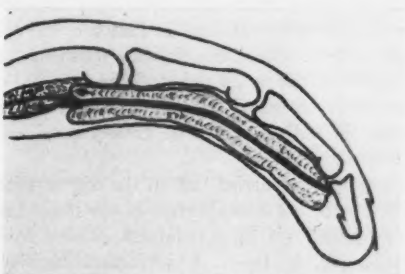

FIG. 32. Purulent tenosynovitis flexor sheath of index, mid or ring finger. Schematically showing tendon sheath markedly distended with pus. Unless promptly drained, this pressure will cause ischemic necrosis of the flexor tendons and will rupture proximally into the deep palmar space.

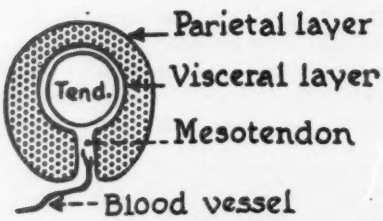

FIG. 33. A cross-section of a sheath enclosed tendon shows the synovial sac which encloses it and also the thin membrane which forms the mesotendon through which pass the blood vessels nourishing the tendon. (From Mason, M. L.: *Christopher's Textbook of Surgery.*)

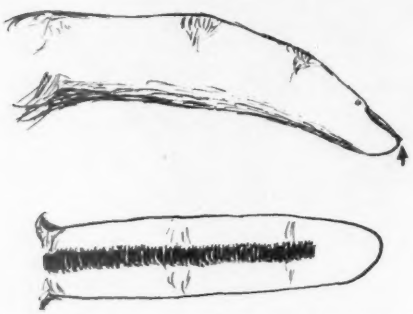

FIG. 34. Tenosynovitis of finger. Kanavels four cardinal signs: (1) finger diffusely swollen; (2) tenderness all along tendon sheath; (3) finger held in position of slight flexion; (4) extension of finger causes severe pain.

became more acute, though it remained localized. Incision and drainage of the distal pulp space were performed by his doctor. However, during the ensuing week the process spread in a fulminating manner.

Examination upon admission to our institution revealed extensive abscess formation of the hand and forearm. The diagnosis was: purulent tenosynovitis of the radial bursa with rupture of pus into the retroflexor, Parona's space. The ulnar bursa did not appear to be grossly involved at this time.

Surgery was carried out on the day of admission. Pus in the distal portion of the radial bursa was drained via an ulnolateral incision in the thumb (fig. 41, *Op-1*). A significant quantity of pus was evacuated from the retroflexor, Parona's space, via the radial forearm incision. The thenar or adductor space, thought to contain pus, was not involved. All overt necrotic tissue was debrided: the distal pulp, the distal segment of the flexor tendon and sheath (fig. 41, *Op-1*).

In the early postoperative period, the acute process subsided promptly, yet drainage continued. Two weeks later, the thumb pulp area required further debridement (fig. 41, *Op-2*). Irrigation revealed free communication between the thumb and forearm incisions. Additional drainage of the ulnar bursa was provided by an ulnar, forearm incision. Postoperatively, drainage continued.

It then became apparent that the continuing deep smoldering infection was being propagated by a substantial amount of retained, necrotic tendon.

Thorough re-exploration was performed on the 30th day (fig. 42, *IIIA*) and revealed necrosis of the index and middle sublimis tendons in the distal forearm, extending into the carpal tunnel (fig. 42, *IIIB*). Additional exposure through a palmar incision showed the finger tendons to be free from disease here (fig. 42, *IIIC*). Partial severance of the transverse carpal ligament per-

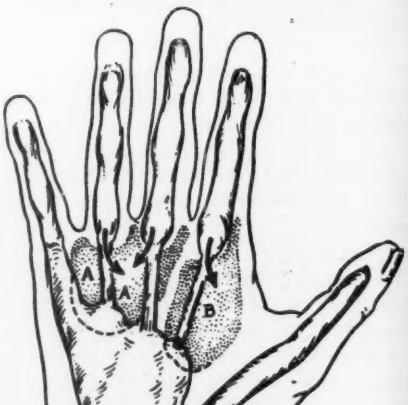

FIG. 35. The spread of infection through the various tendon sheaths and fascial spaces of the palm and forearm shown diagrammatically. (From Mason, M.L.: *Christopher's Textbook of Surgery.*)

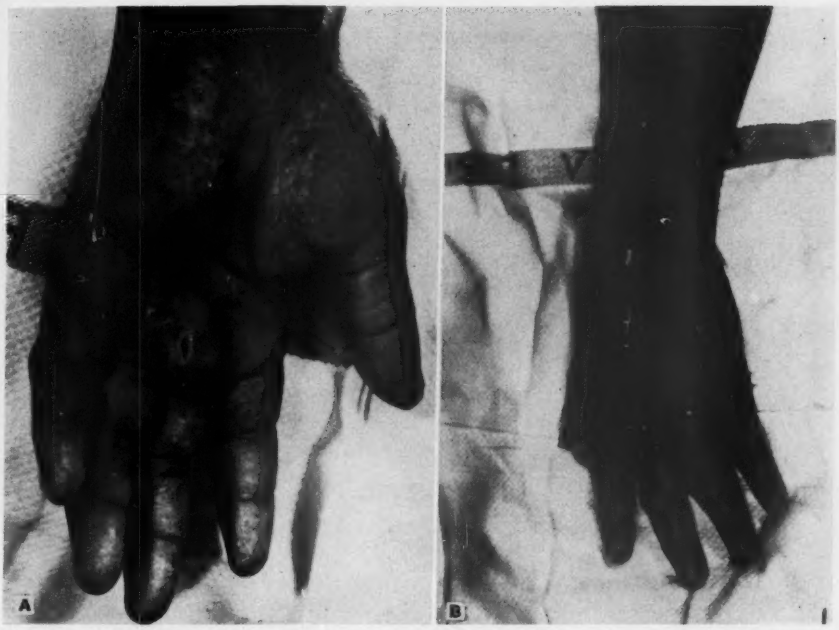

FIG. 36. *A*. (J. B.) Deep, severe infection of hand. Site of origin, volar callus. No significant new trauma. (After 24 hours of soaks and massive antibiotic therapy.) *B*. Marked edema, dorsum of fingers, hand and forearm.

mitted excision of these gangrenous tendons, along with an unattached segment of necrotic thumb tendon.

One week later (5 weeks after admission) at the fourth and final surgical exploration, more necrotic material was debrided, including the palmaris and flexor carpi ulnaris tendons. After this, prompt healing ensued.

During the period of active infection, the hand was maintained in the position of function. Though severely limited, any return of motion during the rehabilitative period will be functional and useful, allowing pinch and grasp (fig. 43).

The protracted course of this patient's infection with progressive tendinous necrosis reminds us again of the prompt need for bold and accurate action.

An earlier and thorough exploration of the forearm, perhaps at initial surgery, might have led to more prompt and complete debridement, less loss of important structures, earlier healing and better return of function. Once gangrene occurs in tendon, thick fascia, cartilage or bone, the infective process will smoulder and drag out for months. The long term immobilization, edema, contracture and fibrosis may result in a painful, frozen, nonfunctioning hand.

Case L. A., a veterinarian, represents a smouldering infection of the hand which was not brought under complete control during our earlier experience with these cases. A deep palmar abscess developed following laceration of skin by a broken test tube (fig. 44*a*, *b*). Prompt surgical exploration revealed pus deep to the palmar fascia but no gross involvement and necrosis of the tendons, sheaths or midpalmar space. After this, chronic infection and drainage persisted for 5 to 6 weeks in spite of the treatment employed: immobilization in the position of function, moist packs and antibiotics (fig. 44*c*, *d*). The patient became dissatisfied and sought help elsewhere.

In retrospect, early surgical re-exploration was indicated along with judicious debridement of all necrotic tissue present.

Case S. T. was referred to us with a protracted, pyrogenic infection in the dorsal wrist area, secondary to moderate injury 9 months before. X-ray revealed carpal bone involvement which

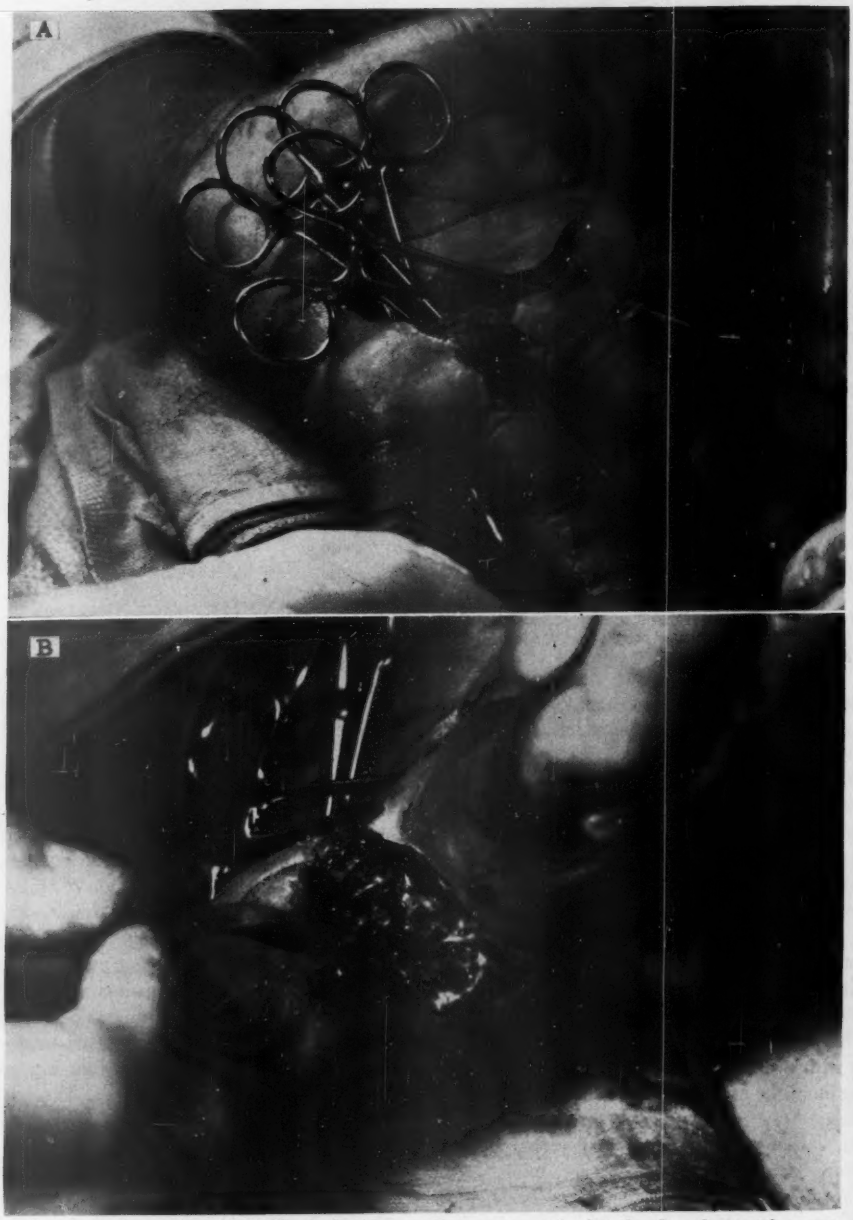

FIG. 37. (J. B. Operation 1). *A*. Necrotic tissue and pus present in distal palm, three web spaces and volar, finger, synovial sheaths. All involved spaces drained and openly necrotic tissue debrided. *B*. Involved web space.

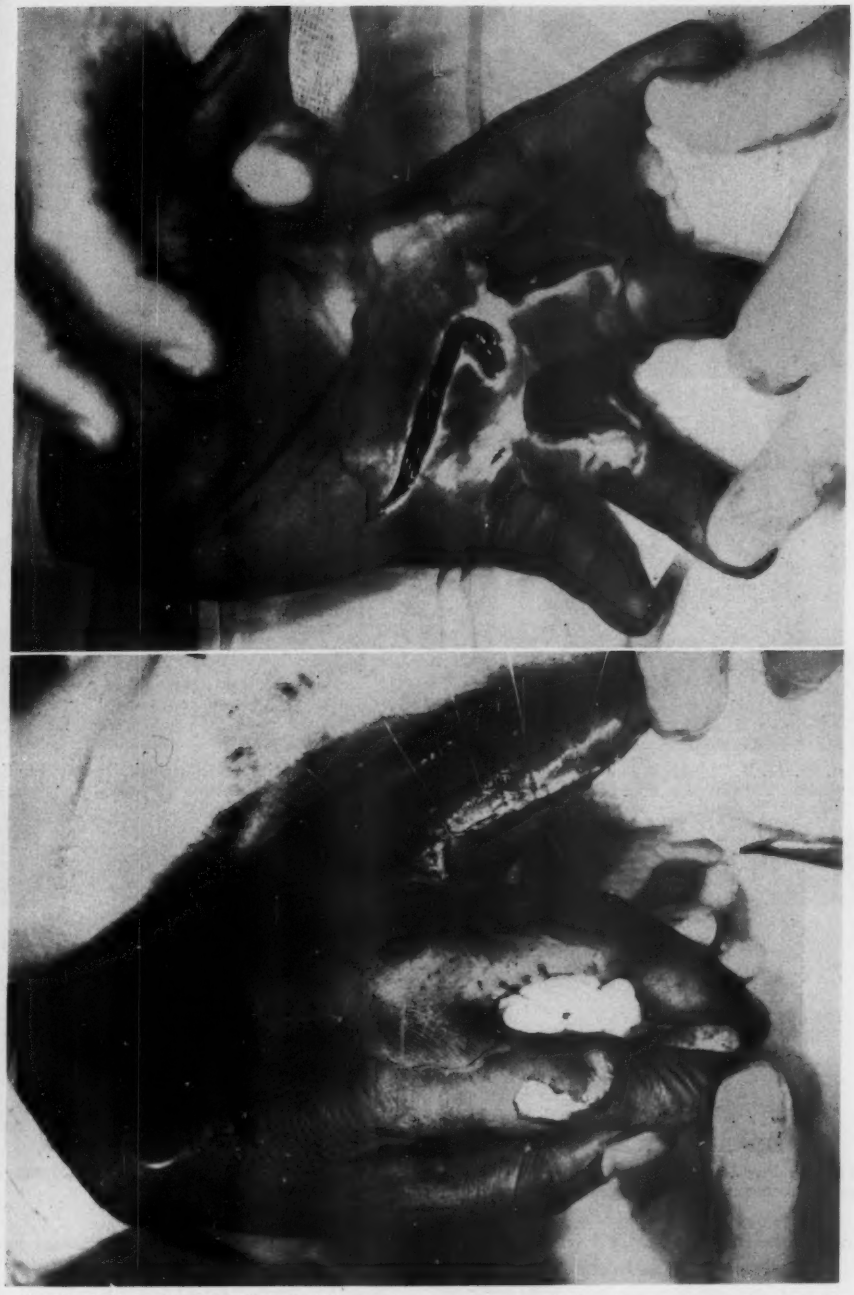

FIG. 38. (J. B. Operation 2, 8th day). All wounds clean and granulating. Large, deep web space defects immediately covered with split skin grafts.

FIG. 39. (J. B.) All wounds healed 1 month later. His range of motion (although limited at this time) included a good pinch, a result possible only when immobilization is performed in position of function. Web space also healed promptly.

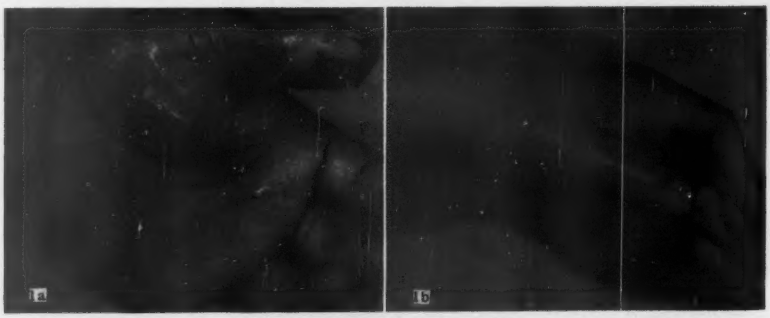

FIG. 40. Purulent tenosynovitis, radial bursa from trivial injury of the thumb pulp (*1a*). Rupture into Parona's forearm space (*1b*). Thenar space swollen, but contained no pus.

proved to be the propagating focus. The wound was promptly explored and debrided. All visible carpal cartilage and necrotic bone were radically excised. Prompt healing ensued followed by a gratifying degree of painless, functional motion (fig. 45*c*, *d*).

Specific chronic granulomatous infection. Case H. H.: Chronic tuberculous tenosynovitis of both

FIG. 41. Prompt incision and drainage of radial bursa and Parona's space. Debridement of necrotic tissue, thumb pulp (*Op-1*). Further debridement required 6 days later (*Op-2*). Purulent drainage persisted from both areas of incision. Ulnar incision revealed pus in ulnar bursa in distal forearm.

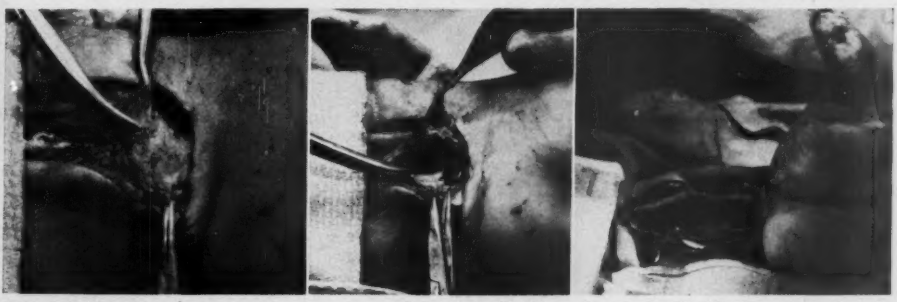

FIG. 42. Infective process still active 1 month later, indicating, deep necrosis. Exploration *via* volar midline. Operation 3: necrotic sublimis tendons of index and midfinger removed (*IIIA, B*). Necrosis extended beneath transverse carpal ligament. At palmar exploration, no necrosis (*IIIC*). Prompt healing ensued.

FIG. 43. Early follow-up (1 month after wounds healed) showing significant limitation of motion and near ability to pinch actively.

forearms (fig. 46). Modest limitation of finger flexion, right; previous surgery, dorsum of left wrist for "ganglion;" prompt recurrence.

The right forearm was explored first, and the characteristic granulomatous involvement of the synovial bursae and sheaths was observed encasing the volar flexor tendons (figs. 47, 48, 50) from the musculotendinous junction, through the carpal tunnel and into the proximal palm. It was completely excised. At a later date, the extensor tendons in the left forearm and wrist were similarly encased. Excision was performed (figs. 49, 50). Characteristically, as in this patient, there is a free plane of cleavage immediately surrounding each tendon which is entered by sharp dissection and permits excision with no damage to the tendons. When the disease process is more advanced, the tendons become invaded and may have to be sacrificed. Late follow-up motion is shown in figure 51.

Case V. S.: Blastomycosis, dorsum of hand (fig. 52). In this patient, the chronic, fungating granuloma disappeared within 6 months after treatment with stilbamidine in January 1955. Residual scarring and chronic ulceration followed. Two years later, when first seen by us, he presented a painful, ulcerated hand with bony destruction in the fifth metacarpal. The fourth and fifth fingers were immobile in extension, at the MP joints especially. He was unable to use this painfully limited hand effectively (fig. 52).

The specific yeast fungi, isolated before drug therapy, were not identified again throughout the long, ulcerated period. All forms of therapy failed. Partial amputation was performed after considerable deliberation, though the patient had been quite willing for several months (fig. 53). The painless and functioning thumb-index finger unit enabled the patient considerably greater independence in self-care with his environment and areas of work and skill.

COMMENTS

In all patients who developed abscess formation and/or necrosis, the process was evident upon admission to our hospital. Pyogenic arthritis and osteomyelitis developed in 2 patients (C. U., S. T.). The functional results were good. In 3 (probably 4) patients, purulent tenosynovitis developed. There was good functional result in case J. B., without tendon loss; necrosis of flexor tendons of the little finger resulted in amputation in case K. I.; necrosis of multiple forearm tendons caused poor function, early in case B. R.; and in case L. A. the result is not known.

Coagulase positive Staphylococcus in pure culture was identified as the initiating organism in all of these acute infections, irrespective of the patient's environment at the time of onset: home, work or hospital. Repeated cultures, in the main, revealed the same organism in pure strain; in only several instances were other organisms cultured simultaneously.

These staphylococci tested were variably sensitive to penicillin, streptomycin and tetracycline, but all were highly sensitive to chloromycetin and erythromycin.

Streptococci were not found in any initial wound culture. In one patient (C. U.) a subse-

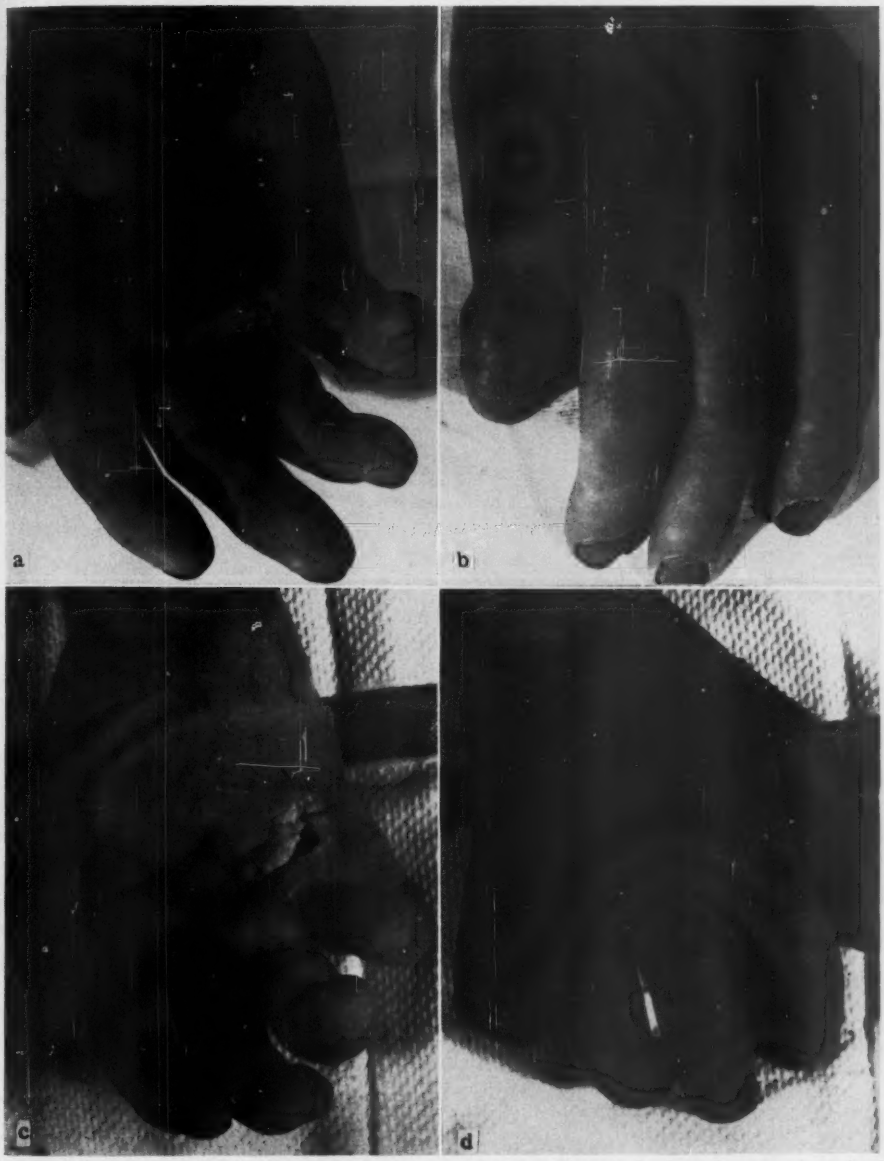

FIG. 44. Infection of distal palm (*a*, *b*) in a veternarian secondary to broken glass puncture (*c*, *d*). Incision and drainage only deep palm tendon sheaths and web spaces explored and drained. The wound failed to heal after 6 to 8 weeks of conservative therapy. The patient sought help elsewhere.

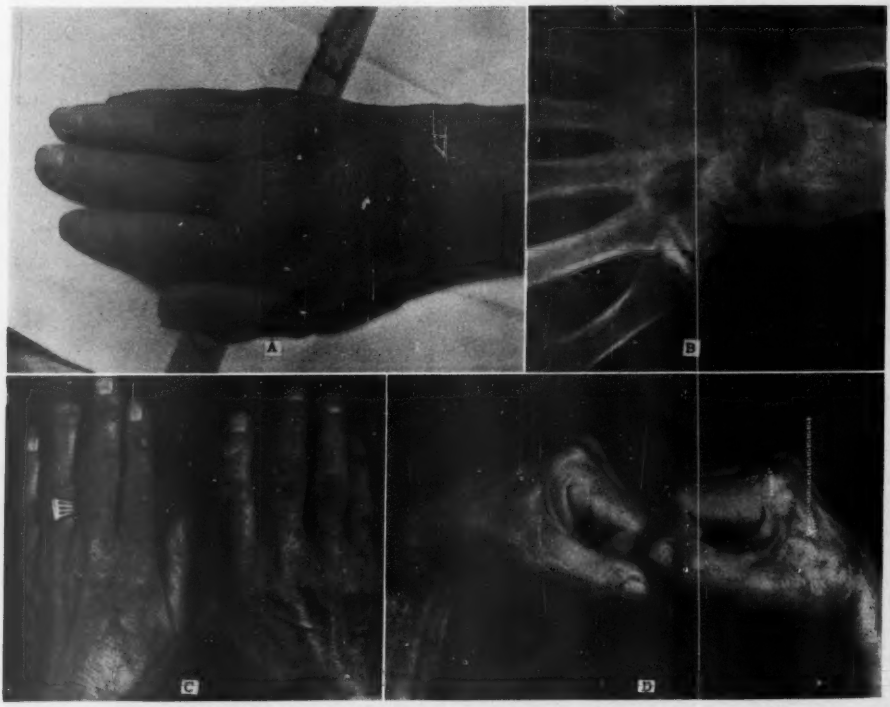

FIG. 45. *A*. Chronically draining unhealed wound resulting from knife injury 9 months previously. Immediate surgery was unsuccessful. The patient now has painful, stiff, swollen wrist, hand and third, fourth and fifth fingers. *B*. X-ray reveals osteomyelitis; necrotic soft tissue, cartilage and bone radically debrided. *C*, *D*. Pain free range of motion of wrist and fingers (6 months follow-up).

quent wound culture revealed a mixed growth of staphylococci and streptococci. The fulminating, spreading infections caused by the Streptococcus and so vividly described by Koch,[5] have not been observed.

This is in marked contrast to earlier reports in which the Streptococcus, in pure or mixed form, caused the great majority of hand infections and serious complications.[3] Flynn[3] and also Aronsson[1] in 1955 reported a significant decline in the incidence of hand infections and their serious sequelae during the previous 15 to 20 years. Both these authors stress the important role of prevention and early and expert treatment. Aronsson found a dramatic decline in the number of hand infections with residual disabilities reported to have occurred during the period 1930 to 1950. The specific decrease during 1933 to 1935 is interesting. Sulfa drugs were available in limited quantities in 1936 and 1937, but not until 1938 were they regularly employed. Both reports consider the antibiotics to have played a role, but perhaps not the major one. The functional fate of our cases likewise appears to be directly proportional to the severity of the infection at the time of our initial examination and the judicious aggressiveness of treatment.

The number of drug resistant strains of Staphylococcus is gradually increasing and may adversely affect the incidence of hand infections and serious complications in the future.

FIG. 46. Tuberculous tensosynovitis of both wrist areas. *Right*, volar; *left*, dorsal. Previous surgery for removal of "ganglion," left dorsal wrist. Some limitation of flexion right.

SUMMARY

An increasing number of patients are hospitalized at our institution with serious hand infections caused by coagulase positive Staphylococcus.

We must be constantly aware of the large number of severe hand infections which are associated with minimal or no trauma. Most of our cases are examples of this.

The behavior of these infections is influenced by the virulence, local action of the infecting organism upon the host tissue and host resistance.

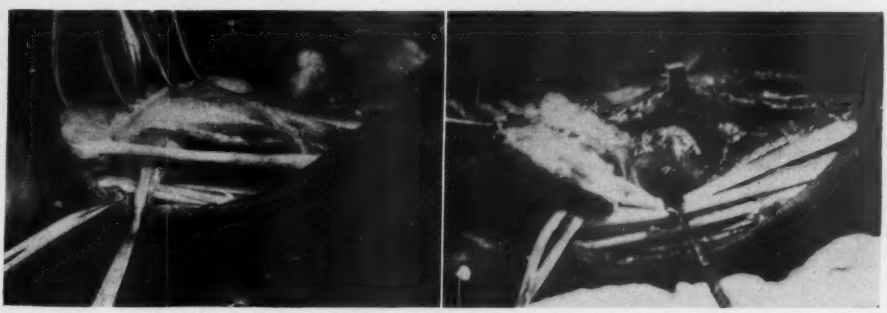

FIG. 47. Surgical excision of granulomatous process. This must be sharply incised to the individually encased tendons. Right forearm.

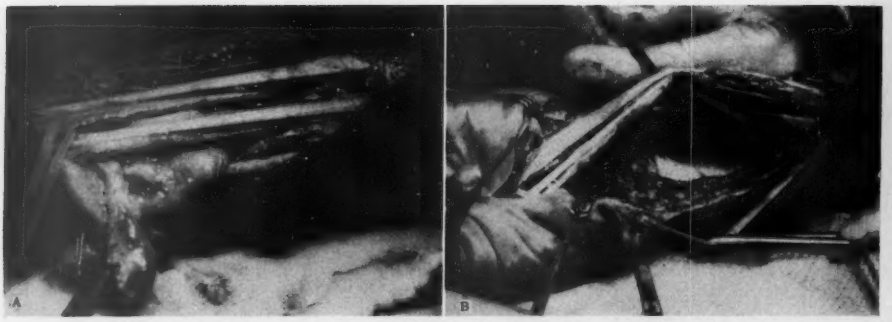

FIG. 48. *A*. Granulomatous tissue involved all flexor tendons from musculotendinous junction to palm. *B*. Transverse carpal ligament incised to gain more exposure.

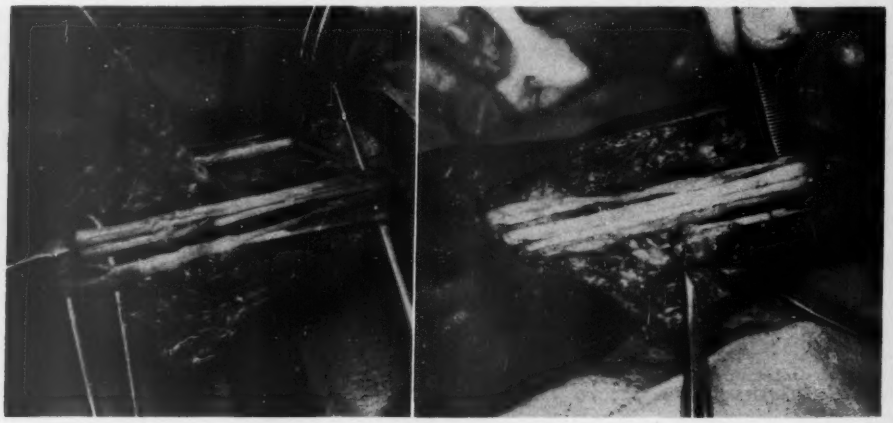

FIG. 49. Excision of chronic tuberculous tissue from extensor tendons, left. Had previous surgery at dorsal wrist level for "ganglion."

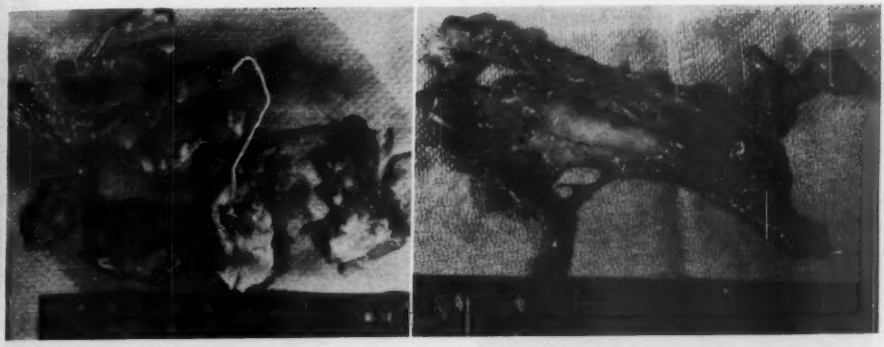

FIG. 50. Excised specimen from both right and left forearms

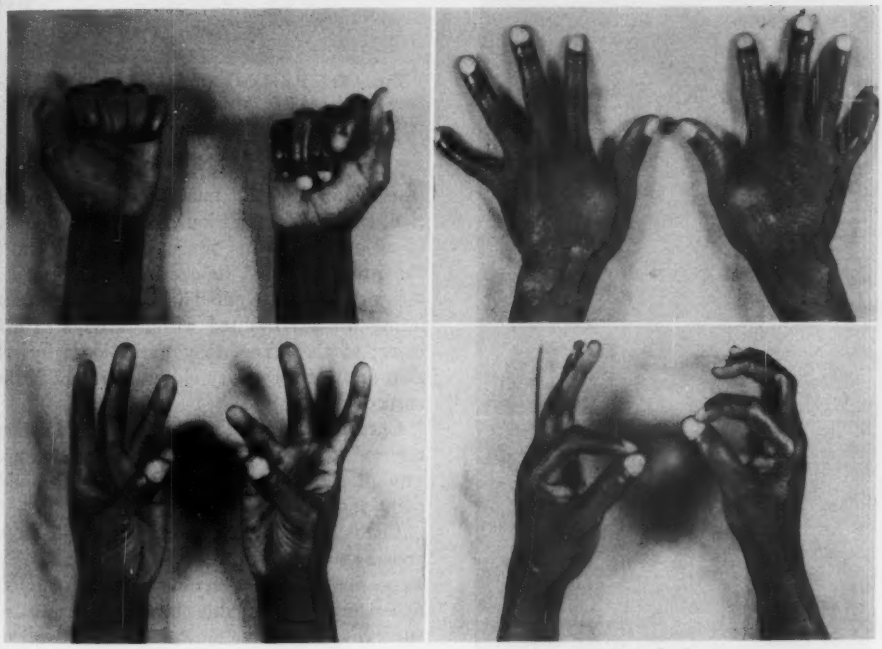

FIG. 51. Follow-up range of motion, 2 years postoperatively

Of especial importance are the influence of the specific anatomical features of the area involved and the necrotizing effect of the Staphylococcus.

Infection often leads to severe limitation of hand function because of the associated pain, stiffness, swelling, joint and ligament contraction and tendon immobility. Thorough knowledge of prevention, early recognition, diagnosis and accurate therapy of these infections are required to regain as near normal function as possible.

The infected hand, when immobilized, must be maintained in the position of function.

Antibiotics, though helpful at onset, will not cure localized pus. It must be drained. In addition, the prompt judicious, radical debridement of all overt, necrotic tissue was carried out in these reported cases. Re-operation and secondary debridement was required in 3 cases. Clean, granulating wounds resulted within 7 days in most cases; at that time, they were closed by split skin graft or secondary closure. Prompt healing ensued.

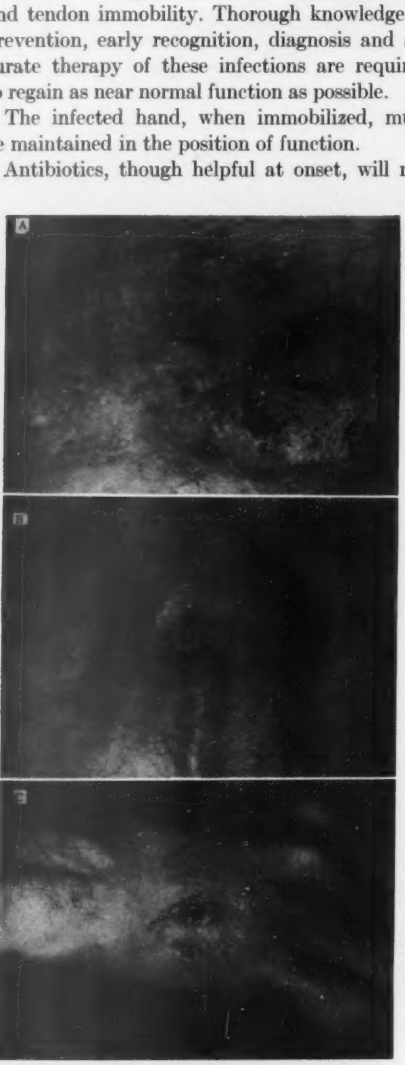

FIG. 52. *A*. Blastomycosis, dorsum right hand. Fulminating, granulomatous process. *B*. Appearance after stilbamidine treatment, June 1955. *C*. Nonspecific ulceration and scarring (1957); wrist, hand, and third, fourth and fifth fingers are painful, stiff and useless.

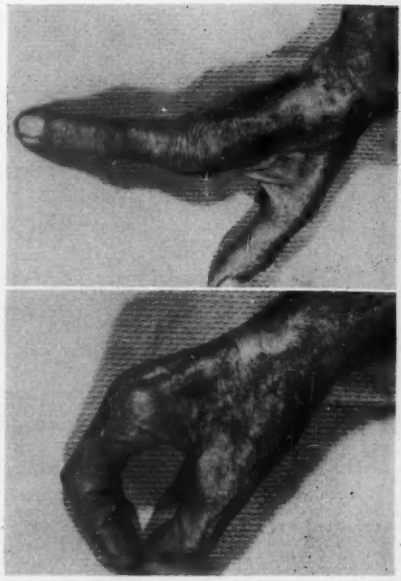

FIG. 53. Painless, functioning thumb-index finger unit (1958) after radical surgery. Middle finger filletted, its good tissue used for dorso-ulnar closure.

Chronic, protracted hand infections, in patients referred to this institution, also responded upon prompt surgical exploration, thorough debridement and early closure.

Vigorous, closely supervised, rehabilitive exercises and treatment must be initiated as quickly as possible. The surgeon must strive to be equally experienced in this vital phase of treatment and supervise it until the greatest range of motion is regained. Complete healing of infected wounds and complete cure of a severe infective process are a small accomplishment unless the patient regains a hand with useful function.

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* Deceased June 1960. Mr. Donald C. Cowley, Acting Manager.

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ARTERIOSCLEROTIC DISEASE OF THE CAROTID ARTERY*

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Every surgeon has observed the problem of arteriosclerosis and noted that it is a destructive process involving primarily the wall of the artery itself. This process progresses to cause occlusive disease of large and small vessels, and secondarily produces vascular insufficiency in the tissue supplied by the artery. Contrary to the early descriptions of arteriosclerosis, it is now common knowledge that it may be a localized process and in some cases is being treated successfully by surgeons everywhere. The exact biochemical and physiologic facts in the production of arteriosclerosis are being studied extensively in an attempt to learn its true etiology and the correct medical treatment. Until this has been solved in the laboratory, direct surgical attack offers the only hope for people with occlusive disease. There is no substitute for blood flow to organs of the body and the mechanical obstruction frequently involved in vascular disease can, at the present time, only be alleviated by operative procedures.

With the use of grafts of various types, normal blood flow can now be re-established in large occluded vessels with relatively low mortality and a high degree of success in reducing the morbidity of the occlusion. The present problem confronting every surgeon is arterial obstruction in a vessel that is technically too small for the use of a graft. In many instances the diseased carotid artery in the neck falls into this category.

It is now suspected that arteriosclerotic occlusion of the internal carotid artery in the neck is one of the most common causes of cerebrovascular accidents. This fact is magnified when it is realized that the disease is in a place readily accessible to surgical operation and that cerebrovascular disease is the third most common cause of death and is probably the most common cause of invalidism today.

This communication proposes to relate the recent advances reported in the literature correlated with personal experience in the diagnosis and surgical treatment of brain insult produced by carotid artery insufficiency.

The introduction of anticoagulants, hypotensive drugs, arteriography, vascular surgery, hypothermia, and refined methods of anesthesia has provided a cure for many people who otherwise would have had a fatal or debilitating disease.

HISTORIC EVOLUTION

Verneuil,[1] in 1872, probably reported the first case of thrombosis of the internal carotid artery. This case was caused by a nonpenetrating injury of the neck. The syndrome of spontaneous occlusion of the carotid artery was first described by Chiari[2] in 1905. In 1914, Hunt[3] emphasized the role of the carotid arteries in producing lesions of the brain and urged their routine examination. Moniz[4] with the introduction of arteriography contributed to the ease with which it can be diagnosed, and in 1937 reported 4 cases. In 1951, Johnson and Walker[5] found 101 reported cases and added 6 cases of their own. They stated, "It is interesting that until 1951 there were only 17 cases in the American literature and 85 in the European literature." The records of more than 50 cases have been personally reviewed by the author. One can conclude that this is a common clinical entity that has long been neglected.

ETIOLOGIC FACTORS

By far the most common associated finding of carotid occlusion is arteriosclerosis.[6-10] Other causes are rare, but the next most common factor is thromboangiitis obliterans.[5, 8, 9, 12] Courville[12] has suggested many associated findings such as syphilitic arteritis, anomalous vessels, pressure by tumors or cervical rib, suppurative infections of the neck, aortic aneurysm and embolism, and heart disease. Traumatic factors such as injuries to the neck, basilar skull fractures, surgical injuries and tonsillectomies with postoperative hemorrhage are also mentioned.[13, 14] Hypertension does not usually play a part as the blood pressure is

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TABLE 1

*Incidence of carotid artery disease in a series of 249 routine angiograms on 174 patients**

Detected carotid artery disease		43 patients 51 arteries
Total occlusion	5	
Severe stenosis (60% or more)	12	
Moderate stenosis (30% or more)	14	
Mild stenosis	20	
Bilateral disease	8	

* In each case the neck was shown.

most commonly normal.[5, 15] Retrograde extension from a thrombus in a cerebral vessel or from a bleeding aneurysm has been postulated.[5, 9, 16] Alterations in the walls of blood vessels, stasis, hypercoagulation, and endocrine disturbances may also contribute.[17] Debilitating diseases such as endocarditis, polycythemia vera, periarteritis, and stress may predispose to occlusion.[9, 16, 18] Boldrey[15] feels that atlantoid compression is important in some cases. It should also be mentioned that a physiologic obstruction may exist not confirmed by anatomical findings.[19] This is described in association with increased intracranial pressure and decrease in cerebral blood flow from reflex adjustment centers in the brain. One may conclude that carotid occlusion may be related to many conditions, but is primarily related to arteriosclerosis.

RELATIVE FREQUENCY

Fisher[20] found an incidence of carotid artery disease in 9.5 per cent of routine autopsies. It is interesting that in 218 cases of vascular occlusions of the cerebral blood flow, 41 lesions were in the carotid artery and only 12 occlusions of major vessels adjacent to the circle of Willis were found. In 200 autopsied cases[21] not one instance of occlusion of the middle cerebral artery was found even though this was frequently the clinical diagnosis. The incidence of carotid disease is undoubtedly comparable to that of cerebral hemorrhage and hypertensive encephalomalacia. In a series of 249 angiograms on 174 patients in which the neck was shown, carotid artery disease was found in 43 patients and 51 arteries. Table 1 shows the extent of the disease. The cases of mild stenosis were coincidental and the patients had no symptoms; 56 per cent of these patients had symptoms.

It occurs in the 30-to-60 age group[9, 22] but is also seen in children[23] and the elderly.[22] Sex preponderance is in men 8 to 1.[21, 24] The left side is affected in 65 per cent of the cases;[5] the right in 35 per cent.

PATHOLOGIC PHYSIOLOGY AND ANATOMY

The functional integrity of the cerebral hemispheres is usually related to that of the carotid arteries[12] and collateral circulation. Symptoms are not related to the carotid artery itself but to cerebral ischemia produced by carotid stenosis, thrombosis, or embolus.[10] The syndrome of carotid artery insufficiency is usually that of cerebral ischemia in the distribution of the middle cerebral artery. This is explained by the fact that the most important collateral circulation is from the contralateral side via the anterior communicating artery (fig. 1). This usually provides the majority of blood to the anterior cerebral artery leaving the middle cerebral deficient in blood (fig. 2). Asymptomatic carotid occlusion is made possible by adequate collateral through the anterior communicating, posterior communi-

FIG. 1. Diagrammatic representation of the circle of Willis showing the deficiency of the middle cerebral artery in carotid artery thrombosis.

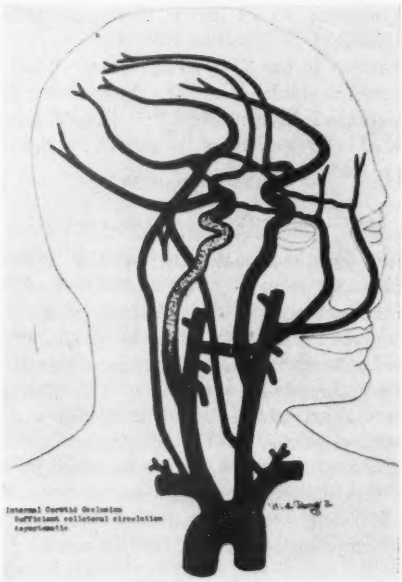

FIG. 2. Diagrammatic representation of asymptomatic carotid artery occlusion made possible by adequate collateral circulation through the anterior communicating, posterior communicating, and ophthalmic arteries.

FIG. 3. Diagrammatic representation of completely insufficient collateral circulation and reason for infarction of entire ipsilateral hemisphere in carotid artery occlusion.

cating, and ophthalmic arteries (fig. 3). In cases of completely insufficient collateral circulation, the entire ipsilateral hemisphere may be infarcted (fig. 4). In unusual cases with a large

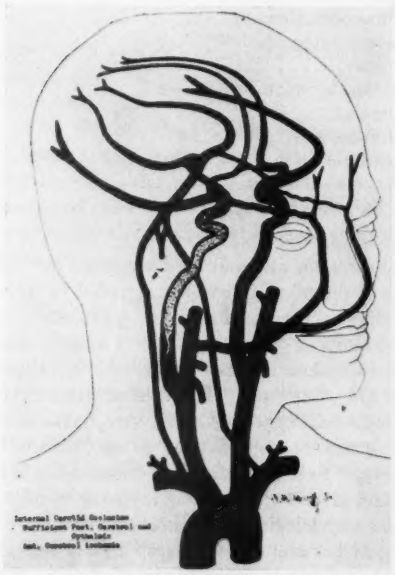

FIG. 4. Diagrammatic representation of deficient collateral circulation to the anterior cerebral artery in carotid artery occlusion.

FIG. 5. The entire hemisphere on the side of carotid occlusion is shown filling from the opposite side through the anterior communicating artery.

posterior communicating and ophthalmic artery and small anterior communicating artery, the arterial deficiency may be in the distribution of the anterior cerebral artery, the symptoms and signs being in the leg (fig. 5). The entire hemisphere on the side of carotid occlusion may be filled from the opposite side through the anterior communicating artery (fig. 6). The cerebral hemisphere may receive the majority of the blood flow from the vertebral arteries in some cases of bilateral carotid insufficiency (fig. 6*A*). The

FIG. 6. Severe bilateral carotid stenosis with the majority of the cerebral blood flow from the vertebral circulation. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

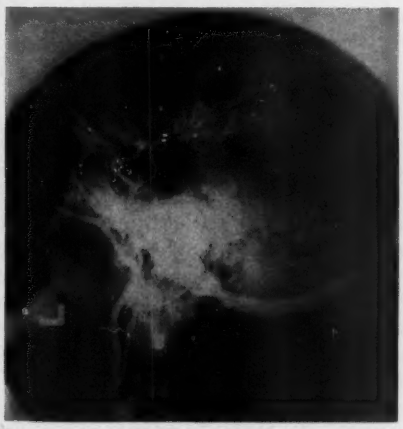

FIG. 6*A*. The cerebral hemisphere receiving blood via the ophthalmic artery in complete internal carotid occlusion. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

FIG. 7. Intracranial obstruction of the middle cerebral artery is shown on the first film.

FIG. 8. The second film taken approximately one second after the film in figure 7 reveals filling in the distribution of the middle cerebral artery from the terminal branches of the anterior cerebral artery.

hemisphere may also receive blood via the ophthalmic artery (figs. 7 and 8). The region of the middle cerebral artery too may receive blood from anastomotic channels with the anterior cerebral artery of the ipsilateral side. Gradually progressive senility to intermittent episodes of stupor may be caused by bilateral carotid stenosis

(figs. 9, 10, and 11). In some cases a fine state of balance in the required carotid blood flow is present and subsequent to re-establishing the normal flow in one carotid artery, the patient may become almost completely clear mentally and have no subsequent episodes of stupor. The intermittent character of the symptoms in the carotid artery syndrome may be explained by the presence of a carotid stenosis with fluctuations in the blood pressure as in shock, associated anoxia, or with multiple emboli from a carotid plaque.[10] Carotid vasospasm may also play a part in such cases.[25]

FIG. 9. Carotid stenosis on one side of a case of bilateral carotid disease. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959).

FIG. 10. Carotid stenosis on the other side in a case of bilateral carotid disease showing post-stenotic dilation.

FIG. 11. Same patient as in figures 9 and 10 after endarterectomy performed on this side. Patient has had no subsequent symptoms of carotid insufficiency. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

PATHOLOGY

The first portion of the internal carotid artery is especially liable to arteriosclerosis. Mehnert[26] found it to be second only to the abdominal aorta as a site of sclerosis. The pathologic picture in the brain is not too clear, but it has been stated[20] that it depends on "the scars of ancient processes, variability of site of occlusion, secondary thrombosis, embolization, collateral circulation, and the size of cerebral softening" (fig. 12). The pathology in the carotid artery is that of an arteriosclerotic plaque.[10] This process is degenerative and destructive and affects the media as well as the intima.

Complete occlusion may be precipitated by superimposition of a thrombus on the already marked stenosis or there may be hemorrhage into the plaque. Frequently, the opposite side and other cerebral vessels show no evidence of the arteriosclerotic process[21] (fig. 13). There may be a complete occlusion in the neck, and an occlusion from an embolus to an intracranial vessel (fig. 14). Thus far, it is not possible to determine the presence of intracranial obstruction if there is also an obstruction in the neck preoperatively.

SYMPTOMATOLOGY

The symptomatology may be varied and obscure as one would conclude from the pathologic

FIG. 12. Arteriosclerotic plaque removed from the internal carotid artery by thromboendarterectomy.

FIG. 13. Complete carotid occlusion in the neck. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

physiology of carotid insufficiency. Gurdjian and Webster[16] described 4 methods of clinical onset: (1) explosive scintillating stroke, (2) slowly progressive signs and symptoms characterized by remission and recurrence, (3) the preceding, plus visual symptoms including transient blindness, and (4) cases without signs or symptoms. Johnson and Walker[5] in their extensive review place the onset in 3 categories and found that in 35 per cent of the reported cases there was a sudden catastrophic onset, in 25 per cent a slowly progressive course, and in 40 per cent a history of transient attacks characterized by headaches, hemiparesis, paresthesia, and aphasia. Headache is present in one-half of the cases and may be unilateral and above the eye. The most common disturbance by far is a hemiparesis in 80 per cent of the cases. Characteristically this is more severe in the arm. Aphasia or speech disturbances are seen in 60 per cent of the cases. Paresthesia, convulsions and mental disturbances are not uncommon. In 42 of 107 cases of Johnson and Walker[5] there were some eye symptoms, the most common being transitory episodes of unilateral blindness. A homonymous hemianopsia is present in 10 per cent of the cases. Shapiro[9] has described head noises as a soft roaring sound and sensation of escaping steam in the ear. Bilateral carotid disease should be suspected in all cases of senility.[10] The symptomatology is varied, but the diagnosis can be suspected. It must always be proved, however.

PHYSICAL FINDINGS

The clinical findings are usually related to ischemia of a certain part of the brain and not to the carotid artery in the neck. There may be a complete lack of pulsation in one carotid artery in the neck or pharynx. This is a reliable sign when consistent with the clinical picture but unfortunately a transmitted pulsation is frequently felt. A thrill may be felt or a bruit may be heard over the bifurcation when a stenosis is

FIG. 14. Same case as in figure 13 after carotid thromboendarterectomy revealing intracranial obstruction of the middle cerebral artery. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

present and is a significant sign. Compression of the opposite carotid may produce weakness or syncope.[16] The ophthalmodynamometer is used to measure the blood pressure in the eye and is useful in cases of constant insufficiency of the carotid artery.[27, 28] The pressure is decreased in the ipsilateral eye.

FIG. 15. Intracranial aneurysm in patient with subarachnoid hemorrhage.

FIG. 16. Opposite side of patient in figure 15 showing carotid stenosis.

FIG. 17. Case with obstruction intracranially simulating an obstruction in the neck.

FIG. 18. Same case as in figure 17 showing upward movement of dye in film taken one second later.

DIAGNOSIS BY ARTERIOGRAPHY

With few exceptions every patient who has had a cerebrovascular lesion should have bilateral carotid arteriograms as soon as possible unless he is moribund.[10] The neck, including the bifurcation of the carotid should always be shown.[10] This is particularly true if any surgery on the carotid vessels in the neck or head is contemplated. Figure 15 reveals an intracranial aneurysm on one side and figure 16 reveals a carotid stenosis on the other side in the same patient. Surgery on either side without this knowledge might be fatal.

Another finding that should be noted is that of an intracranial obstruction simulating an obstruction in the neck[10] (fig. 17). The dye may taper off on the dependant wall of the artery and appear to meet an obstruction (fig. 18). On consecutive films, however, the dye will be seen in its slow course to continue upward, the outlet possibly being through the ophthalmic artery.

TREATMENT

Treatment is directed at re-establishing the blood flow to the brain. This is sometimes more difficult than seems apparent. A review of the literature and personal communication with other investigators reveals that the carotid blood flow has been re-established in relatively few cases. Many reported unsuccessful attempts have

TABLE 2

Reported cases in which the author claims to have re-established the carotid blood flow

Author	Year	Cases	Preop Arteriogram	Type surgery	Postop Arteriogram	Results
Eastcott[32]	1954	1	Stenosis	Resection-direct anastomosis	Not done	Improved
Denman[33]	1955	1	Left stenosis Right occlusion later	Resection-arterial graft Resection-graft common to external carotid	Patent Patent	Improved Improved
Arnstein[12]	1955	10	Complete block Stenosis Complete block Stenosis Not given Not given Not given Not given Not given Not given	Thromboendarterectomy Thromboendarterectomy Thromboendarterectomy Thromboendarterectomy Thromboendarterectomy Thromboendarterectomy Thromboendarterectomy Thromboendarterectomy Thromboendarterectomy Thromboendarterectomy	Not done Not done Not done Not done Not done Not done Not done Not done Not done Not done	Improved Not improved Improved Improved for 6 months Not given Not given Not given Not given Not given Not given
Perlow[34]	1955	1	Complete block	Thrombectomy	Carotid open, middle cerebral blocked	Expired in 2 days
Carrea[35]	1955	1	Stenosis	External to internal anastomosis	Patent	Improved
Cooley[36]	1956	1	Stenosis	Thromboendarterectomy	Not done	Improved
Lin[37]	1956	1	Stenosis	Resection and vein graft	Patent	Improved
Edwards[38]	1956	1	Stenosis	Resection and direct anastomosis	Not done	Improved
Galbraith[39]	1957	5	Stenosis Complete block Complete block Complete block Stenosis	Nylon graft bypass from subclavian to internal carotid artery	Not done	Improved
Toole[40]	1957	2	Complete block Complete block	Thromboendarterectomy Thromboendarterectomy	Patent Not done	Improved Improved

TABLE 3

Clinical findings and results on patients in whom a retrograde flow was obtained and endarterectomy performed

Name	Age	Sex	Clinical	Preop Arteriogram	Postop Arteriogram	Results
L. B.	75	F	Hemiparesis, aphasia	Bilateral stenosis, more on right	Patent	Much improved, no aphasia, some weakness
E .F. H.	65	M	Hemiparesis	High grade stenosis, left	Patent	Much improved, minimal aphasia, little weakness
W. M.	65	M	Hemiparesis	Marked stenosis, left; occlusion, right	Patent	Marked improvement, no weakness
T. W.	50	M	Hemiparesis, aphasia	Marked stenosis, right; occlusion, left	Patent	Marked improvement, little aphasia, little weakness
M. E. D.	16	M	Hemiparesis, aphasia, coma	Occlusion, left	Patent, middle cerebral blocked	Little improvement
S. J. S.	71	M	Hemiparesis, aphasia, coma	Occlusion, right	Patent, middle cerebral blocked	Little improvement
B. J.	41	M	Hemiparesis, aphasia	Marked stenosis, left	Patent, blocked at siphon	Little improvement
D. W.	67	M	Semicoma	Bilateral stenosis	Patent	Marked improvement
R. B.	59	M	Hemiparesis, aphasia	Bilateral stenosis	Patent	Little improvement
M. L. B.	67	F	Stupor, hemiparesis	Bilateral stenosis	Patent	Expired in 2 days, cause unknown; artery open at autopsy

been made. These attempts include thromboendarterectomy,[29] arterectomy,[30] thrombectomy,[31] common carotid-jugular vein anastomosis,[3, 7, 17] temporal muscle grafts up to the surface of the brain,[17] and cervical sympathectomy[9, 16, 17, 18] (table 2). The first reported case in which it was claimed to have re-established the carotid blood flow was in 1954.[22] Since that time there have been approximately 24 cases reported, but in only 6 of these was it proved by postoperative arteriography. This is very important in evaluating surgical results on the carotid artery. One case has been personally followed in which the patient had a complete carotid occlusion and refused surgery. In a few hours he made a complete recovery and has remained normal for 6 months on anticoagulants. Of the 24 cases in which carotid flow was reported re-established, the surgical procedure was thromboendarterectomy in 13 cases, resection and direct anastomosis in 2 cases, resection and arterial graft in 1 case, thrombectomy in 1 case, external to internal carotid anastomosis in 1 case, resection and vein graft in 1 case, and bypass procedure with nylon graft—5 cases.

In 10 cases at the Baptist Memorial Hospital,

Memphis, Tennessee (table 3) the carotid circulation has been re-established by thromboendarterectomy and proved by postoperative arteriography 1 month after operation in 8 cases, 10 days after operation in 1 case, and 1 day after operation in 1 case. In 3 additional cases in which no anticoagulants were used, the artery thrombosed, and in 1 other case the artery proved to be occluded in the head.

INDICATIONS FOR OPERATION

The primary objectives of this operation are:[10] (1) to prevent thrombosis and embolism, and (2) to relieve ischemia. The diagnosis should be made and surgery performed during the stage of transitory symptoms before permanent brain damage has occurred. Operation is indicated in (1) cases of complete occlusion or severe stenosis with transitory symptoms, (2) cases of complete occlusion or severe stenosis with emboli to smaller vessels from which the patient may be expected to recover, (3) cases of complete occlusion or severe stenosis with early mental deterioration, and (4) bilateral disease with early mental deterioration and/or transitory symptoms.

FIG. 19. Exposure of the carotid artery and intraluminal clot usually seen distal to the plaque at the bifurcation. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

FIG. 20. The technique of removing the arteriosclerotic plaque from the carotid artery is shown. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

Although it is unlikely that clot removal can be successful after a week, operation is not contraindicated because this has not been determined with certainty.

TECHNIQUE OF THROMBOENDARTERECTOMY[10]

Thromboendarterectomy has the advantage of being simple and can be performed in a few minutes. The operation is performed under local anesthesia so the neurologic status of the patient can be observed at all times. A shunt is kept ready in case the patient's condition gets worse

after a stenosed artery is clamped off. This problem is not encountered in cases of complete occlusion (fig. 19). The artery is exposed and a clamp is placed on the proximal portion of the common carotid artery and external carotid artery distally. Heparin is always injected into the artery when a clamp is used and flow stopped. The internal carotid is then opened distal to the plaque and almost invariably a clot will be found. In some cases this is blown out by retrograde flow from the head, and in other cases it is necessary to apply suction into the artery. If no retrograde flow can be obtained the operation is abandoned (fig. 20). If a retrograde flow is obtained, a clamp is placed on the distal internal carotid and thromboendarterectomy is performed (fig. 21).

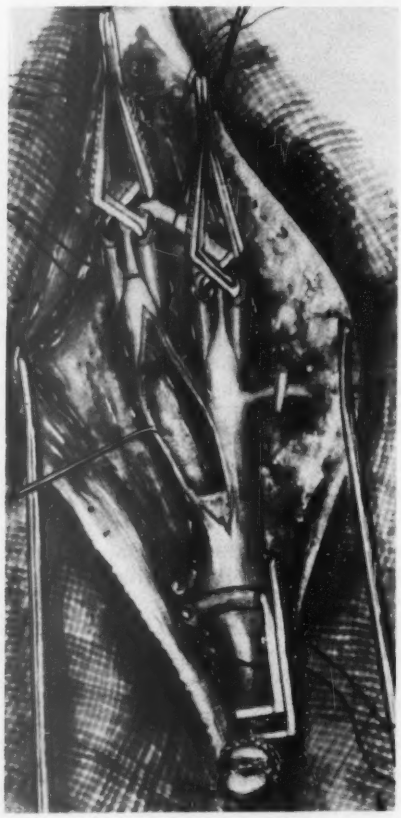

FIG. 21. The intraluminal area is shown subsequent to thromboendarterectomy. Note the simple silk suture through the distal intima attaching it to the wall. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

FIG. 22. The closure of the artery performed in the usual manner. The artery is irrigated with heparin just prior to the last stitch. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

After the plaque is removed the distal intima is sewed to the wall by two or three silk sutures (fig. 22). The artery is then closed with arterial silk, the patient being started on intravenous heparin at this time. This is continued until the long-acting anticoagulants are in effect. An example of the results are seen in figures 23 and 24.

SUMMARY AND CONCLUSIONS

1. Carotid arterial occlusive disease is common and should always be considered in any patient with symptoms of a stroke, convulsions, dizziness, syncope, or other more bizarre neurologic pictures.

FIG. 23. Severe stenosis in internal carotid artery preoperatively. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

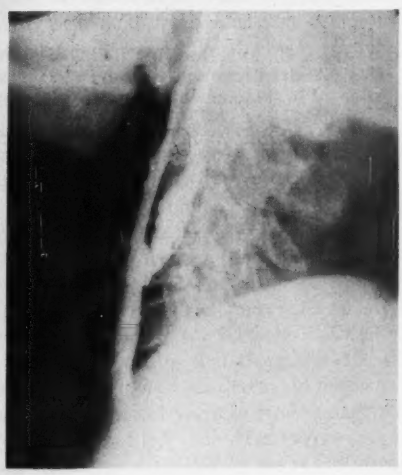

FIG. 24. Internal carotid artery seen (fig. 23) one month after thromboendarterectomy. (From Murphy, F. and Miller, J.: J. Neurosurg., *16:* 1, 1959.)

2. Such patients should have carotid angiography, and if carotid occlusion is found, thromboendarterectomy should be immediately carried out.

3. The procedure of thromboendarterectomy has to date produced the best results and is a simple, safe procedure if properly done.

4. Ten cases in which the above procedure was done have been personally followed by the author, and examples of such cases are presented.

5. Recognition of the frequency of the disease, study of the anatomy of cerebral circulation, understanding of the pathology, physiology, and proper application of treatment will, it is predicted, make cerebral vascular surgery a new and dramatic advance in medicine.

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THE BULBOCAVERNOSUS FAT PAD TRANSPLANT FOR SEVERE STRESS INCONTINENCE AND VESICOVAGINAL FISTULA: RATIONALE OF THE PROCEDURE, INDICATIONS AND TECHNIQUE*

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Involuntary leakage of urine in the female whether because of stress incontinence or vesicovaginal fistula presents a problem to many physicians at some time during their practice of medicine. The patient and her physician become aware of its presence almost immediately. Once a leakage of urine has occurred, the patient can no longer engage in her normal activities as she must constantly be on the vigil for the telltale spot on her dress or be frequently changing a near saturated perineal pad to prevent detection of her cryptic and remorseful problem.

The usual causes of stress incontinence are related to obstetric trauma or, in the postmenopausal patient, this condition may be due to decreased blood flow in the venous plexus around the bladder. Some authors[1, 6] have expressed the view that the primary lesion is a descent and funneling of the neck of the bladder with loss of the normal posterior urethrovesical angles. A vesicovaginal fistula may occur or recur as a result of obstetric trauma, pelvic surgery (for benign or malignant diseases), irradiation for malignant pelvic diseases, chemical injuries to the urinary bladder from potassium permanganate vaginal burns, or trauma from automobile accidents with injuries to the pelvis and bladder.

One author[15] states that there are at least 75 methods which have been devised in the course of an equal number of years to cure urinary stress incontinence in the female. Many types of operations have been described for stress incontinence and all have demonstrated their merits. Most of these procedures are simply modifications of basic operations performed early in the development of gynecologic surgery and, although most of them have enjoyed a period of popularity, many have been replaced by newer techniques.

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Reconstruction or replacement of weakened paraurethral structures is the object of every operation for stress incontinence. Hodgkinson[7] has demonstrated the urethrographic technique which indicates the importance of the vesicourethral angle of the urethra. The purpose of all these techniques to prevent the escape of urine is to provide external pressure on the urethra as a substitute for the normal sphincter mechanism which has been destroyed.

The process of controlled micturition requires a proper balance between the propulsive forces of the bladder and urethral sphincteric restraint. The latter is missing in the case of stress incontinence, whereas in vesicovaginal fistula gravity plays the dominant role.

With the almost universal acceptance and performance of the total rather than the subtotal hysterectomy, the number of urologic injuries in the course of gynecologic surgery has increased greatly in the last few years. Many surgical procedures, varying from the usual vaginal plastic anterior repair of Kelly[8] to the Millin fascial sling procedure[14], have been devised for the treatment of stress incontinence. Although the usual vesicovaginal fistula responds to a simple plastic procedure approach, the recurrent fistula or the very large fistulous tract presents a perplexing problem.

No single surgical procedure so far devised has proved to be an infallible cure for this common distressing ailment of dribbling of urine from the urethra or the leaking of urine from an unphysiologic hiatus in the bladder.

The bulbocavernosus fat pad transplant as described by Martius,[12] and recently re-emphasized by McCall,[10] is a procedure with diversified indications and is justifiably used for the very large fistulous tract, the recurring fistula or severe stress incontinence.

RATIONALE

The choice of surgical technique for the relief of stress incontinence involves four factors.[2]

These are: (1) the severity of the incontinence; (2) the extent of concomitant trauma to the musculofascial supports of the bladder, as evidenced by cystocele; (3) the hormonal status of the patient—whether she is pre- or postmenopausal; and (4) previous surgery which has failed to afford sufficient relief.

The vesicovaginal fistula presents another problem. The operator should remember that the tissues around the fistula often have a poor blood supply, and that the wound itself is inevitably contaminated with urine. The very large fistula and the recurrent fistula demand respect and often tax the imaginative intuition of the pelvic surgeon. It is now felt that one procedure is available for all the above discussed problems. The bulbocavernosus fat pad transplant accomplishes these four objectives: (1) adds support to the urethral neck; (2) adds bulk to the bladder neck and base of the bladder; (3) decreases and/or obliterates the dead space between the vaginal mucosa and the bladder; and (4) brings in a new blood supply to an area of diminished vascularity. The first three objectives are indicated for the unusually severe cases of stress incontinence, whereas the last three objectives play important roles in prevention of the recurrence of the fistulous tract. If a failure should occur, as in the case of the vesicovaginal fistula, very little has been lost since the other bulbocavernosus fat pad can be utilized. This type of vaginal surgery does no harm if another type of procedure should be selected, either vaginal or abdominal.

The supravesical approach of Marshall and associates[11] is not interfered with if the bulbocavernosus procedure is performed. One need not have the fear of pressure necrosis of the urethra or bladder neck with this procedure, although it could occur with the fascial sling type procedure for stress incontinence.

In the case of the large vesicovaginal fistula or the recurrent fistula the bulbocavernosus fat pad transplant, if not entirely curative on the first procedure, will decrease the size of the fistulous tract; the other fat pad can be used without difficulty to cover the remaining defect in the bladder with curative effect. Usually the one operation is sufficient to stop all involuntary leaking of urine, whether by vesicovaginal fistula or severe stress incontinence.

This pelvic surgical procedure seems physiologic and it has a tremendous place in the treatment of the recurrent or very large vesicovaginal fistula. It is much better than the surgical diversion type procedure, after one failure at attempted closure.

The main indications for the bulbocavernosus fat pad transplant are severe stress incontinence, recurrent stress incontinence after surgical corrective failure, and the large or recurrent vesicovaginal fistula. When the simple anterior vaginal plastic wall repair for severe stress incontinence is not sufficient, especially where there is sagging of the neck of the bladder, supplementary plastic surgery is often necessary to lift and support the bladder neck. The smooth fat flap support lifts the bladder neck and replaces the cavernous tissue, and the fat pad transplant is carried out just the same.

ILLUSTRATIVE CASES

Severe stress incontinence and marked cystocele. A 45-year-old gravida 7, para 7, white woman came to the Gynecology Clinic with a history of "something falling from her vagina," and of having recurrent bladder infections and progressive and severe stress incontinence since her last delivery 8 years before. Pelvic examination revealed a third degree cystocele, and a second degree prolapse of the main pelvic structures. In view of her multiparity and recurrent genitourinary infections with progressive stress incontinence, it was decided to do an elective vaginal hysterectomy and anterior plastic vaginal repair. It was noted that there was marked sagging of the bladder neck and marked redundancy of the vaginal mucosa over the extreme prolapsing bladder. It was decided to do the bulbocavernosus fat flap transplant to add support and bulk to the vesicourethral angle. The procedure was carried out in the manner described by Martius. Repeated examinations have shown no recurrence of the cystocele and the urinary stream infections have not returned. It is now about 30 months since the operation and the patient is having no difficulties in passing her urine, nor has she had any recurrence of the stress incontinence.

Recurrent stress incontinence with bladder neck laceration. A 78-year-old gravida 2, para 2, white woman came to the gynecologic clinic because for the past 10 to 15 years she had had a progressive loss of urine when she coughed, sneezed and laughed. The last menstrual period had been some 32 years before. Examination on straining revealed easily the marked overflow and mild cystocele. She had dilation and curettage and a vaginal hysterectomy with the usual anterior and posterior vaginal

plastic repairs. The postoperative course was uneventful for about 2 to 3 months and then the stress incontinence recurred and there was noted sagging of the bladder neck. The cystocele had not recurred but the stress incontinence became progressively worse.

In view of the previous procedure it was elected to do a fascial sling procedure, starting from the vaginal approach with the urethra being freed for the rubber strip guide.

Inadvertently in the vaginal dissection, the bladder neck was torn and urine immediately drained through the hiatus. Because of this new problem, the sling procedure was abandoned, and it was decided to do a bulbocavernosus fat pad transplant. The urethrovesical tear was approximately 1.5 cm. in transverse length and the laceration was closed with interrupted small catgut sutures. The bulbocavernosus fat flap was used with the right pad in the prescribed manner. It is now about 2 years since the operation and there has been no occurrence of a vesicovaginal fistula nor a return of the stress incontinence.

Comment. The usual closure of a fistula is sufficient. The operative success is assured if the sutures can be placed in two or three layers without tension and if no hollow space is allowed to remain between the sutures of the fistula and the vaginal mucosa. The bulbocavernosus fat flap is interposed as a pad to this empty space. The effect of the bulbocavernosus fat flap in securing fistulous sutures and its effect as a plug in dead spaces have been demonstrated by Martius.[12]

The fat pad pedicle contains the blood vessels and the nerves to the musculature and these are preserved. Martius[10] emphasized the importance of using a thick fat flap and not the bulbocavernosus muscle in transplantation. It is of particular value because it provides an excellent pad and support. The bulbocavernosus fat pad transplant is utilized as a protective filling when the suture line needs support and padding.

Large and recurrent fistula. A 40-year-old gravida 4, para 4, white woman was first seen in this Clinic on April 5, 1959, with a history of a brownish vaginal discharge for 2 months. The discharge occurred after and before two menstrual periods and had continued until the next menses. Examination showed an essentially normal pelvis and the cervix was described as clean. Because of the patient's age and the history of unusual bleeding, a Papanicolaou cytologic smear was taken and sent to the pathologist. The smear was reported as positive for malignancy and suggestive of squamous carcinoma. A cold knife conization of the cervix and an intrauterine curettement were performed under general anesthesia. The pathology report was of early well differentiated epidermoid carcinoma *in situ* of the cervix with no clearly demonstrated infiltration. The endometrium was in the late secretory phase.

Since the woman was 40 years of age and desired to have no further offspring, it was elected to do a total abdominal hysterectomy. While the bladder was being stripped off anteriorly from the uterus a 2-cm. laceration occurred near the dome of the bladder. The laceration was noted and closed with the usual technique of 3 layers. Some 12 days later urine appeared in the vagina and a diagnosis of vesicovaginal fistula high in the vaginal apex was made. The routine procedures for location were used. An abdominal approach was used to close the fistulous tract, and a suprapubic counter drainage was used. The fistula recurred on the 18th day, and was demonstrated by methylene blue injected into the bladder and soaking the vaginal sponge.

When the patient was seen in consultation, it was found that the fistulous tract originated high in the apex of the vagina, and cystoscopic examination revealed the fistula to be large and about 3 cm. in diameter. The stage was nearly set for the urinary diversion type procedure, but it was suggested that this patient might benefit from the bulbocavernosus fat flap transplant.

The patient was given cortisone preoperatively, as described by Collins,[4] and prophylactic sulfonamides. The fat flap transplant procedure was performed on August 5, 1959, and the large fistulous tract was dissected free and the fistula closed under tension. The fat pad flap was placed in the position as suggested by Martius and sutured in place. The patient was continued on the prophylactic sulfonamides and also given intravenous estrogens and ascorbic acid to promote vascularity and healing of the pelvic structures. Some 4½ months after the operation there was a recurrence of the fistula. On examination, no fistulous tract could be demonstrated vaginally but there was urine in the vagina and the methylene blue test was positive. Cystoscopic examination revealed a 0.5 by 0.2-cm. elongated fistulous hiatus in the vesical mucosa. The patient was again given preoperative cortisone and prophylactic sulfonamides and then she was reoperated upon on February 8, 1960. The small "funnel" shaped fistulous tract was dissected free and the mucosa was closed without tension. The left bulbocavernosus fat pad was dissected free (the right pad previously had been utilized), and its posterior

attachment was left attached to preserve the blood and nerve supply. The fat pad was then brought over the suture line and sutured in place. The indwelling Foley catheter was removed on the 20th day and the patient is about 1 year postoperative with no recurrence of the fistula nor any evidence of stress incontinence.

Comment. In all cases where closure of the fistula is done under tension, the bulbocavernosus fat pad transplant is the procedure of choice. This surgical operation is also valuable in the large fistulas where little tissue is available, resulting in closure under tension, as in this last illustrative example, and creation of a dead space between the fistula line and the vaginal mucosa.

TECHNIQUE

The bulbocavernosus fat pad transplant procedure of Martius[12] commences as a simple anterior vaginal plastic procedure. The vaginal mucosa is incised close to the urethra, in the midline, toward the vaginal cuff apex for a distance of approximately 5 to 6 cm. The fistula (if this is the indication) is dissected free, the fistulous edges are freshened and sutured without tension, if possible, with fine catgut sutures. The right or left labium majus is palpated with two fingers, one inside the vaginal incision and the other outside the labium majus. A fat pad transplant is formed from one labium majus (this is about as thick as the thumb and long as the finger). A ridge of thickness of a finger breadth is clearly perceived which contains the bulbocavernosus muscle and vestibular bulb. An incision of the skin is made in the ridge over the labium

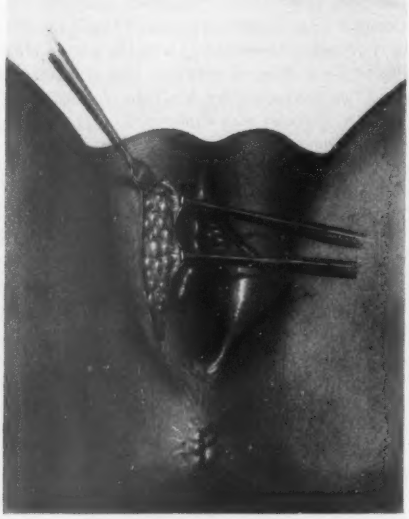

FIG. 1

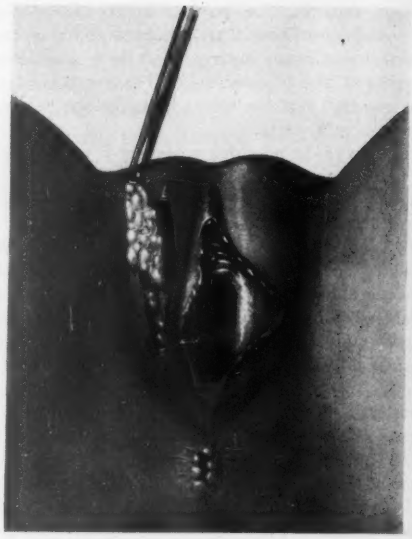

FIG. 2

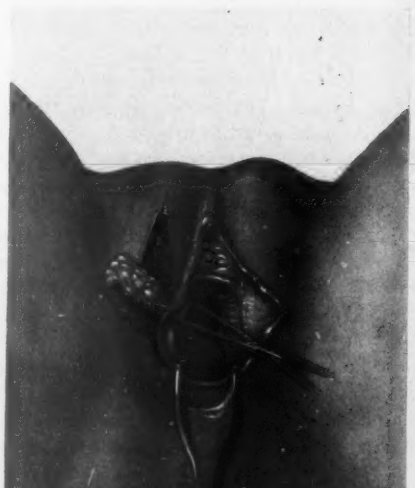

FIG. 3

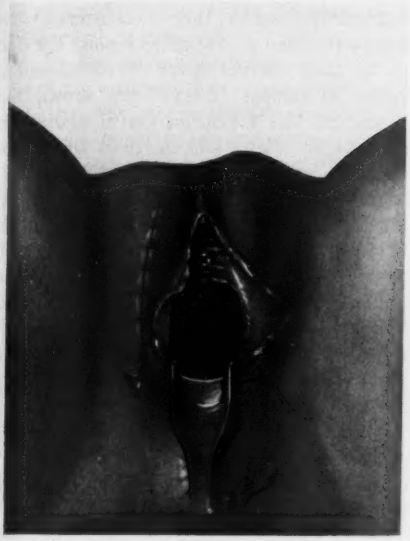

FIG. 4

majus on the medial aspect (fig. 1). A fat flap pad transplant is formed and left attached to the pedicle posteriorly (fig. 2). The fatty tissue flap is dissected with a few strokes by sharp dissection. This flap contains the portion of the bulbocavernosus muscle which was palpated. The bleeding vessels are ligated.

The skin between the vaginal incision and the fat pad graft is tunnelled by blunt dissection. The fat pad transplant, with the flap remaining attached posteriorly, is drawn through the tunnel (fig. 3) and placed under the vesicourethral neck and/or over the suture line of the fistula. The free end of the flap is sutured to the left side of the vaginal incisional angle (if the right fat pad is utilized) as high as possible. One or two moderate sized catgut sutures are usually sufficient.

The vaginal wound is closed in the usual manner with small catgut. A rubber strip drain is placed in the labial incision from the upper pole to a protruding position from the lower pole (fig. 4). The labial incision is closed with small catgut sutures. The drain is advanced on the first and second postoperative days and removed on the third postoperative day.

COMMENT

This manuscript serves to re-emphasize the applicability of a surgical operation which has many merits. This procedure can be used with excellent results as attested by Martius[12] and von Massenbach[16] in large series in which this technique was used. This vaginal plastic operation has been shown to have diversified indications ranging from severe stress incontinence to the large or recurring vesicovaginal fistula. The control of urination requires a proper balance between the forces of bladder propulsion and urethral restraint. In stress incontinence this balance has been disturbed. Once the uncontrolled escape of urine has been positively established whether it be after delivery, pelvic surgery or trauma, the next step is to determine the exact location and extent of the damage. A thorough pelvic examination, cystoscopic survey of the urinary bladder and intravenous urograms are essential for the proper diagnosis and treatment to localize the lesion whether near the trigone, dome of the bladder or bladder neck.

Stress incontinence is not to be belittled because it may be a constant source of embarrassment to the victim. Many pelvic surgeons see the mild to moderate cases of stress overflow and these are usually associated with pelvic relaxation and a moderate degree of cystocele. These patients usually respond surgically to the routine anterior vaginal plastic repairs of Kelly.[8] When the patients have severe stress incontinence, this may present a perplexing problem. If the incontinence is not alleviated by the usual simple plastic repair, another procedure must be contemplated. Case 1 is illustrative of severe stress incontinence with marked pelvic relaxation and a third degree cystocele. Rather than risk the possibility of a failure of the simple plastic vaginal repair, due to the marked sagging of the neck of the bladder, it was elected to do the bulbocavernosus fat flap transplant to add sufficient bulk and support to the bladder neck, and the operative procedure had excellent results.

Some of the causes of vesicovaginal fistula are hematoma, infection, and concealed perforation of the bladder. Injudicious placement of sutures or an area of the bladder caught by sutures may become ischemic and slough, resulting in fistulous tract occurrence. Other factors that contribute to the etiology of vesicovaginal fistula are bleeding, use of too many hemostats, lack of exposure and irradiation by physicians who have occasional cases for treatment.

Since the increasing use of the automobile.

with more highway travel and the alarming increase in automobile accidents, more trauma to the pelvis will occur. Many times the bladder will be lacerated and, unless suspected, it may become fistulous. Bone may perforate the bladder and hematoma and/or infection may ensue.

At the Charity Hospital in New Orleans, many young Negro girls are seen with potassium permanganate vaginal burns after an attempt to abort themselves. There is a common folklore that potassium permanganate tablets will cause the girls to bleed and this is interpreted as a miscarriage. In actuality, the bleeding is a vaginal mucosal erosive process causing profuse bleeding by the chemical eroding into an artery, vein or venous plexus. It is conceivable that this substance could erode into the bladder if placed superficially in the vagina and a fistulous tract could result.

Fistulas of obstetric origin have been decreasing, and the probable reasons for this are the excellence in obstetric training and improved facilities for parturition.

As a rule, bladder injuries which occur during abdominal operations involve the dome of the bladder. They are usually recognized and can be safely repaired. Rarely do they complicate the postoperative course, but case 3 is an exception to this statement. The same may be said for most of the bladder injuries which occur during vaginal operations, except for those injuries which involve the urethra and neck of the bladder. The second case is of this type, with the bladder neck lacerated or torn in the process of attempting a fascial sling procedure. This case represents a failure at an attempt to correct severe stress incontinence in an older woman with a marked lack of estrogens in the vaginal mucous membranes, by the usual anterior vaginal plastic technique. In view of the bladder neck tear and the recent failure, it was elected to do the bulbocavernosus fat pad transplant. This added bulk to the bladder neck and brought in the new blood supply to an area of diminished vascularity, and added bulk to the sagging urethral neck. The partial dead space under the vaginal mucous membranes has been obliterated. These injuries are more difficult to repair and the additional procedure of bulbocavernosus fat flap gives more assurance toward total healing and additional support. Imperfect wound healing may lead to an occasional fistula or to stress incontinence of urine.

Many gynecologists urge a waiting period of 5 to 6 months from the time of occurrence of the fistula to the time of attempted repair. They believe this time interval allows the edema and induration to subside. Ragan[13] has shown that cortisone inhibits the formation of granulation tissue and fibroplasia and increases the growth of tissue macrophages. Collins[4] now administers a total dose of 3 gm. of cortisone in a 10-day period of time to all patients with vesicovaginal fistula. Because of the anti-inflammatory properties of cortisone there was noticable decrease in the induration which was present in nearly all of Collins' cases of vesicovaginal fistula. Patient 3 had preoperative cortisone, and marked decrease in induration was found at the time of surgery. Since the patient continued to have leakage of urine (some of the fistulas heal spontaneously after cortisone therapy), operation was scheduled for the next day and the repair of the fistula undertaken. At the time of pelvic surgery the induration had diminished or disappeared and dissection of the fistulous tract with closure was carried out with greater ease and facility. Even with the anti-inflammatory properties of cortisone, the possibility of the spread of infection by contaminated urine still existed. Consequently, the administration of cortisone and the simultaneous administration of prophylactic sulfonamides, penicillin or broad spectrum antibiotics prevented infection of the area by the vaginal contaminates or by the urine. Other systemic diseases, especially diabetes mellitus, should be ruled out before the administration of cortisone.

After surgery, an indwelling catheter was allowed to remain in place in all patients, but no counterdrainage was employed. In the case of the recurrent fistula (case 3), the catheter was left in place for 1 month and the bladder was irrigated with a dilute solution of potassium permanganate (1:10,000 solution), and occasionally with a 0.5 per cent solution of silver nitrate to help prevent formation of granulation tissue in the suture line area.

If the vesicovaginal fistula is easy of access and if surrounded by soft, vascular tissue, it stands a better chance of healing. The operator should be well experienced in vaginal plastic surgery, and if the fistula is not a simple type fistula, there should be no hesitation in placing the patient in the hands of a pelvic surgeon who has had experience in this very specialized work. Success in fistula repair demands of the surgeon good visual acuity, respect for tissue, gentleness

of touch, and an unwavering attention to the minute details of technique.

Usually the genitourinary specialist thinks in terms of the transvesical approach because of his familiarity with this method of operating. The gynecologist prefers even more strongly perhaps the vaginal route. It might be thought that a high vaginal fistula after total abdominal hysterectomy is unsuitable for treatment from below, but good exposure can almost always be obtained by making a generous Schuchardt incision or episiotomy. If the injury is recognized and repaired, sequelae are rare, even though some of the difficulties may occur in the extensive or radical hysterectomy operation.

The preoperative and postoperative use of estrogens given to elderly women with fistula appears logical as a means of improving the blood supply to the vaginal mucous membranes.[5] Oral and intravenous estrogens have been used in the cases that have come under my immediate care or consultation.

If the bulbocavernosus fat pad transplant operation is not successful, or not entirely successful, no great harm has been done and further repair can still be contemplated. This cannot be said with equal truth of the abdominal or transvesical operations. There is no shortening of the vagina with the bulbocavernosus fat pad transplant, such as has been attributed to the partial colpocleisis of Latzko.[9] Therefore, there will be no difficulty in coitus.

As might be supposed, a referred case will often present features of unusual difficulty. Some of the patients have already been subjected to previous operations, and a few are seen as "last resort" cases. This is the type of operation which, at a price, may give relief but which is not a cure. Case 3 was a near last resort type case, having already been operated on twice and now having a large fistulous tract present. Transplantation of the ureters into the rectum, sigmoid or isolated ileal segment described by Bricker[3] is the last resort type of procedure. Although the foregoing genitourologic procedure has a definite place in pelvic surgery, less drastic operations, if curative, should be contemplated.

Previous operations undoubtedly make successful closure more difficult but it is surprising how, with adequate exposure, vascularity of tissue and careful technique, scar tissue can be excised and sound union obtained. Size of the fistula and closure under tension govern the type of operative closure. Although a simple fistula may be successfully closed by many different methods, and stress incontinence eliminated by many operative procedures, the real test for any technique is its reliability or adequacy in the difficult or complicated case.

This procedure may not be the panacea for curing both the vesicovaginal fistula and stress incontinence, but it definitely has a place in the armamentarium of the gynecologist in pelvic surgery.

Probably the most sincere patient in her gratitude is the woman whose involuntary loss of urine has been eliminated and who has been restored to her rightful activity in the midst of her family and social intimates.

SUMMARY

1. Rationale, indications and technique of the bulbocavernosus fat pad transplant have been presented.

2. The problems of the large or recurrent vesicovaginal fistula and severe stress incontinence have been discussed.

3. The bulbocavernosus fat flap transplant has a definite place in the armamentarium of the pelvic surgeon in the surgical treatment of severe stress incontinence and the large or recurrent vesicovaginal fistula.

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CONGENITAL ATRESIA OF THE EXTRAHEPATIC BILE DUCTS: REPORT OF A CASE WITH SUCCESSFUL SURGICAL CORRECTION*

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Seventy years ago Thomson,[1] from a study of autopsy material, first suggested the condition of congenital atresia of the bile ducts. Forty-five years ago Holmes[2] proposed the possibility of surgical correction, and estimated that 16 per cent of those afflicted with congenital atresia were potentially operable. In 1928 Ladd[3] reported the first successful surgical correction of this condition. Schnug[4] in 1958 reviewed the literature and found 39 reported cures including his own case. In the case which we shall present the atresia was correctable and the patient appears entirely well 3 years after the operation.

There are two well supported theories as to the cause of biliary atresia. Yllpö[5] in 1913 suggested that during the phase in fetal development of the proliferation into solid cords of many areas of the alimentary tube, there are arrests and, during the next phase of rapid growth and elongation, a failure to recanalize. The result is atresia of all or part of the biliary system. This explanation is supported by Gross[6] and Arey[7].

The other theory, proposed by Rolleston and Hayne[8] and supported by more recent work on hepatitis by Ahrens, and associates[9], and others[10], suggests that toxins are carried across the placental barrier injuring the fetal liver with resulting cirrhosis or descending cholangitis with fibrotic obliteration of the biliary tree.

The diagnosis of biliary atresia can be made only at operation. The suspicion of this condition leads to diagnosis by exclusion. Prehepatic jaundice ordinarily can be diagnosed and usually excluded.

1. Icterus neonatorum, the commonest form of jaundice, is resolved by the 10th to 14th day. Bile is present in the brown stool and no excess bilirubin spills over into the golden urine. Liver function tests are normal.

2. Congenital hemolytic icterus is diagnosed by the excess fragility of the red cells. A family history of hemolytic jaundice is often obtained.

3. In erythroblastosis fetalis jaundice appears early, from birth to a few hours after, and the Coombs tests and Rh or ABO incompatibility are reliably diagnostic.

4. Syphilis, although now rare, is diagnosed by serology determinations, confirmed by x-ray of the long bones.

5. Septicemia may not have the usual fever but leukocytosis creates the suspicion and blood cultures confirm the diagnosis.

Posthepatic jaundice presents the problem which is resolved most often by surgery.

The inspissated bile syndrome caused by erythroblastosis or of unknown cause, possibly viral hepatitis, may present the same picture as congenital atresia of the bile ducts. The atresia may be extrahepatic and amenable to surgical correction or intrahepatic and inoperable.

In these groups the bilirubin is elevated with a marked increase in the direct reading bilirubin. The jaundice appears slowly and may be first noticed as late as the fifth or sixth week of life. In hepatitis the bilirubin remains elevated or falls over a period of two to six weeks. A slow rise in blood bilirubin is usual in atresia. Liver function tests are normal in most of the two groups as are the proteins, cholesterol and flocculation tests.

Delay in operation is urged by those who have noted an increased operative mortality in the hepatitis patients subjected to exploration.[10] However, delay is criticized by many who have reported a series of cases subjected to operation. The latter note that biliary cirrhosis has often advanced to an irreversible state in many otherwise curable cases, and that 65 per cent are atresia cases, 15 per cent hepatitis, and the rest of undetermined or rare causes.[12] Since the largest number are cases of atresia, the best results will be obtained from early exploration which should not be beyond the second month of life.

Preparation for exploration should first include the realization that the extrahepatic ductal system may be tiny and easily destroyed. Cases are reported in which no bile ducts could be found but the patient completely recovered. Second,

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facilities for cholangiography, polyethylene tubing of 2- to 5-mm. diameter for stents in performing anastomosis must be at hand during surgery. Third, preparation must be made for aspiration with fine needles and the return injection of saline to dilate the gall bladder and bile ducts. The operation should expose the gall bladder area, the hilum of the liver, and the hepatoduodenal ligament. The gall bladder, if found, is aspirated for identification and then, through a tiny mushroom catheter or polyethylene tube tied into the gall bladder, a cholangiogram is made. If this demonstrates a normal system, the tube is left for irrigation and the operation is terminated. If this discloses apparent atresia and no bile-containing duct can be visualized without dissection, a deep biopsy is made in each lobe of the liver and the operation is terminated. Tissue section report of hepatitis means no further surgery but a diagnosis of biliary cirrhosis demands re-exploration, a careful dissection of the hepatoduodenal ligament with anastomosis of any bile containing structure to the duodenum, stomach, or jejunum, in that order of preference. The polyethylene stents, less than 5 mm. long, are used to thread the duct to maintain the patency of the anastomosis.

CASE REPORT

C. U. was a full term, white, male child born on September 20, 1956. He had a normal delivery and weighed 8 lb., 8 oz. The mother received no medications by injection during pregnancy and the baby received no injections. Four siblings were living and well. The mother, aged 25, had no previous history of serious illness. The father, aged 35, gave a history of occasional jaundice and an enlarged liver.

The infant, at 6 weeks of age, was re-admitted to the hospital of birth, Kansas City General Hospital, on October 28, 1956, with a history of 3 days of noticeable yellow skin and sclera and 24 hours of repeated vomiting. He was ill, apathetic and moderately jaundiced with moderate scleral icterus. The liver was 5 cm. and the spleen was 2 cm. below the costal margin and both felt soft, rubbery and smooth. No petechial markings were present and neurologic findings were normal. The stools were chalky and the urine dark and staining. Urobilinogen in the stool was less than 0.1 mg. per cent in repeated examinations. Urobilinogen in the urine was 1 mg. per 1000 cc. Bilirubin determinations rose during 20 preoperative hospitalization days from 7.2 to 20.4 total with direct 8.8 mg. and indirect 11.6 mg. The liver profile showed cephalin cholesterol floccuation, negative; thymol turbidity, 3.5 units (normal 0.5 to 5.5 units); thymol floccuation, negative; alkaline phosphatase, 2.7 Bodansky units (normal 0.5 to 5.5); total protein, 6.15; albumin, 3.45; globulin, 2.70; cholesterol, 325 mg. per cent; esters, 131 mg. per cent (37.2 per cent); red blood count, 4,020,000; hematocrit, 32 per cent; hemoglobin, 12.5 gm.; fragility, less than control; serology, negative.

Operation. The patient was anesthetized with cyclopropane closed circuit with an infant cycle. A transverse incision was used to give wide exposure. The liver was large, tense, rubbery and greenish black with marked lobulation. The spleen was only moderately enlarged but firm. Minimal dissection of the hepatoduodenal ligament exposed an intact pair of hepatic ducts entering a common duct ending in a bulb just beyond the cystic duct. The cystic duct and gall bladder were intact. This represents the most frequent of the potentially curable group in the large series reported by Gross. A hypodermic needle placed in the bulbous end of this common duct remnant revealed the presence of dark green bile. The duodenum was freely movable and not over 5 mm. distant. A primary anastomosis, two layer with a first layer of mucosa to mucosa was made, and a widely patent anastomosis (over 5 mm.) was accomplished without the use of a stent. The small gall bladder was not aspirated, nor was the preparation for operative cholangiography employed. The abdomen was closed by layers with a drain to the anastomosis site.

The postoperative course was uneventful. Brown stools were first noted on the 5th postoperative day. The patient was dismissed on the 17th postoperative day with bilirubin of 2.8 mg. per cent.

At the age of 2, on May 18, 1959, the patient was re-admitted to the hospital jaundiced. Total serum bilirubin rose to 18. Fever of 102° and rapid clearing of jaundice led to the diagnosis of "ascending cholangitis." At this time the liver and spleen were not palpable.

Since this time the patient has continued normal growth and has remained in good health. His present appearance is good.

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PROBLEMS INVOLVED IN EXTERNAL COOLING TO CARDIAC CESSATION*

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As more and more dependable adjuncts to the field of surgery are devised, surgeons find themselves attacking once insurmountable problems with relative impunity. One such valuable adjunct which has achieved prominence, and no little definition, in the past several years is general hypothermia. By artificially decreasing the temperature of an individual, one is able to decrease that individual's metabolic activity and demands to a point at which deprivation of metabolic essentials for prolonged periods of time is not injurious. The successful application of hypothermia in surgery, notably in cardiovascular and neurosurgical fields, is well known.

Current techniques of general hypothermia are divided into two major groups. The first group consists of those techniques involving use of extracorporeal circulation. Venous blood is taken from the patient into a closed circuit in which it is cooled and then pumped actively into the patient's arterial circulation, after having passed through an oxygenating apparatus.

Use of extracorporeal heat exchange makes possible rapid induction to the hypothermic state as well as rapid return to normothermia.[1] In addition, finite control of temperature levels of the patient may be established.[1] Extracorporeal pumping augments the decreased cardiac output secondary to hypothermia as well as serves as a means of organ perfusion during periods of inflow occlusion. Appearance of acidosis during periods of moderate to deep hypothermia[2] is apparently lessened or even aborted by uninterrupted systemic perfusion.

Conversely, damage to the formed elements of the blood, which is attendant to the use of any present extracorporeal unit, obviates the use of this technique over prolonged periods of time as in some attenuated neurosurgical procedures. Heat exchange offers more technical hazards and requires an increased number of trained personnel in its successful use. Recent evidence shows that formation of microbubbles on rewarming by this technique may pose further problems.[3]

The other large category of techniques of hypothermia includes those methods which employ external environmental cooling. Such methods include the use of cooling blankets, corporeal immersion in cold water and others.

In inducing hypothermia by external environmental cooling, one may extend the cooled state for longer periods without fear of the damage to blood elements that accompanies extracorporeal circulation. Fewer technical problems arise with this method, and fewer trained personnel are necessary to carry out "passive"[4] hypothermia. Thus, such a technique may be applied in an area where equipment and technical assistance are not readily available.

Many of the disadvantages of "passive," or external environmental cooling have been well delineated.[1] Slow, cumbersome induction and rewarming are inherent in this method; inadequate control of the patient's temperature and tendency to "drift" are well known. Use of passive cooling without support of extracorporeal perfusion can result in inadequate cardiac output, acidosis, electrolyte aberrations, peripheral sludging, and other undesirable phenomena.

Because of the inability to control adequately the patient's temperature and the tendency to downward drift in passive cooling, this method can make manifest a severe complication of hypothermia: the markedly increased incidence of cardiac irregularities and fibrillation in the hypothermic state at temperatures less than 28° C.

Yet because of the occasional desirability of prolonged cooling and the impracticality of heat exchange in some areas, and in an attempt to more clearly define, scientifically, currently amorphous "hibernation," we feel it is necessary to provide a consistently adequate method for

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external environmental hypothermia. Therefore, the work presented in this paper is an attempt to establish, experimentally, a consistent means utilizing external environmental cooling by which an individual may be taken to levels of, or very near, cardiac standstill and still be returned to a state of normothermia and viability.

METHODS

Thirty-three mongrel dogs, of the average weight of 10.0 kg., were anesthetized with cyclopropane-oxygen mixtures, intubated, and placed on to-fro absorption apparatus. In the first group of 13 dogs, mepazine,* a phenothiazine derivative, was given intravenously in divided doses during early cooling with the total dosage averaging 30 mg. per kg. The second group (20 dogs) received mepazine, 25 mg. per kg., diluted in a continuous intravenous infusion of 75 to 150 cc. of 5 per cent glucose solution throughout early cooling. The last 18 received also 0.2 mEq. per kg. of potassium chloride in the same infusion, completed before an esophageal temperature of 26° C. was reached.

Cooling was accomplished by immersion of the dog in a 50 per cent ice-water mixture; a separate ice pack was placed around the animal's head. At 26°C., cyclopropane was discontinued, and the animal was slightly hyperventilated with 100 per cent oxygen. As circulation became progressively more ineffective, ventilation was proportionately decreased, then stopped. Right-sided thoracotomy was performed during a temperature range from 28° to 26°C., followed by placement of tapes around the superior and inferior venae cava to prevent, both during cooling and rewarming, cardiac distention by regulating venous inflow The azygous was ligated routinely.

Cooling was continued until ineffective cardiac output and/or cardiac standstill took place, in most instances between 16° and 21°C. The dog was then removed from the ice bath and, in the first 17 dogs, rewarming was accomplished by perfusion of the right chest cavity and pericardium with a warm isotonic saline solution. The last 16 dogs were rewarmed by immersion in a warm water-bath. Ten of this group received a 5 mg. total of epinephrine intravenously during rewarming.

* (*N*-methyl-3-piperidyl-)methylphenothiazine supplied as Pacatal by Warner Chilcott.

RESULTS

In 28 of the 33 animals used, effective cardiac action with sinus rhythm was restored during the rewarming phase; 15 (45.4 per cent) of the 33 survived at least 24 hours postoperatively.

In 13 animals (39.4 per cent), ventricular fibrillation occurred during some phase of the cooling. This occurred in almost 60 per cent of the group who received only mepazine, but only in 30 per cent of the group who received both mepazine and potassium chloride. Nine animals demonstrated ventricular fibrillation during the rewarming periods; 7 cases occurred during use of direct intrathoracic instillation of warmed saline. Only 2 cases of ventricular fibrillation occurred in the group that was subjected to warm water immersion.

The use of caval tapes with which to regulate venous inflow during cooling and rewarming was considered to be of significant importance. In 3 of the 5 dogs in whom sinus rhythm was not restored, notable dilation of the heart was observed after failure to regulate inflow. These demonstrated a flabby, very hypotonic myocardium which was unable to contract effectively.

The influence of potassium chloride in preventing excessively low pH values was evident. In all animals receiving only mepazine, blood pH during deep cooling fell to levels of 7.15 to 7.05 or lower. With the addition of potassium chloride to the infusion, the pH at cardiac standstill and shortly thereafter was maintained above 7.3 with only rare exceptions.

At the time of this writing, we are carrying out subsequent experiments with passive hypothermia, with the use of the biologic pressor substance, angiotensin,† in conjunction with mepazine in a 5 per cent glucose intravenous infusion throughout the cooling and rewarming periods. We have been quite impressed, in our preliminary study, with the maintenance of adequate cardiac output to within 5 to 10 seconds of complete cardiac asystole, followed by a prompt return of sinus rhythm and effective cardiac output within several minutes after beginning rewarming. These preliminary studies bear careful follow-up but seem to indicate that angiotensin may provide some answers to at least several problems compounding the use of deep hypothermia.

† The angiotensin used in this study was supplied by Ciba Pharmaceuticals, Inc., as Hypertensin-Ciba.

SUMMARY

The need is emphasized for a consistently adequate method for external environmental hypothermia, both as a current surgical adjunct and as a tool in delineating, or even reproducing safely, that vague entity, hibernation.

An experimental method is described by which, utilizing external environmental cooling, dogs may be relatively consistently taken to levels of cardiac standstill and returned to a state of normothermia and viability.

A promising pilot study, utilizing angiotensin with hypothermia, is mentioned.

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THE OPEN REPAIR OF MITRAL INSUFFICIENCY*

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The mitral valve becomes insufficient when the functional leaflet tissue is smaller in area than the valve orifice. Generally, this reduction in functioning leaflet tissue is the result of rheumatic fever. In a few instances, rupture of the chordae tendineae allows unrestrained herniation of the involved leaflet tissue into the atrium and produces insufficiency. The rheumatic process produces thickening and fibrosis in the leaflets and chordal attachments. If the chordae become too involved, there is shortening which holds the involved valve area in an open position by retraction against and into the left ventricular cavity. If the rheumatic process has been particularly severe, there is further loss of functional valve tissue by curling and rolling of valve edges, and by calcification. As calcification further limits mobility, insufficiency may be produced simply by fixation of the valve in an open position without actual loss of valve tissue.

In cases where there is little calcification and instead only fibrosis, thickening, and shortening of the valve components, the annulus may become dilated, thus adding to the relative loss of valve tissue.

Many attempts to correct mitral insufficiency by closed techniques have been reported.[1-5] These reports have been characterized by technically difficult procedures and generally unsatisfactory results. As open heart surgery has developed, it is only natural that the problem of mitral insufficiency be approached with an open heart technique. The initial reports of the correction of these technically difficult lesions are encouraging.[6-12]

The purpose of this paper is to report our experience with the open repair of a small group of mitral insufficiency cases.

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SELECTION OF CASES

The signs and symptoms of mitral insufficiency need no elaboration here. A loud, apical, systolic, regurgitant type murmur, occupying most of the interval between the first and second heart sounds, is present. Approximately half of the patients have shown auricular fibrillation. Two patients have had a slight diastolic rumble but this was completely overshadowed by the systolic murmur.

Phonocardiograms have confirmed the intensity of the systolic murmur and have shown murmurs to occupy almost all of the interval between the first and second heart sounds. A definite third heart sound has been present in most victims and indicates the severity of the disease.

Radiography has shown a moderate to marked left atrial enlargement and predominant left ventricular hypertrophy and dilation, in a generally enlarged cardiac silhouette. Evidence of biventricular enlargement has corresponded to the degree of pulmonary hypertension present. Calcification at fluoroscopy should be looked for and usually indicates a more severely damaged valve.

The electrocardiograms have been those of left ventricular hypertrophy and overload pattern, except when pulmonary hypertension has been severe, at which time right ventricular hypertrophy has been predominant.

Right and left cardiac catheterization has not been done where the diagnosis has been felt to be quite firm on clinical grounds, and where there was no reason to suspect aortic valve disease. Pulmonary artery and pulmonary wedge pressure determinations have been helpful in deciding for or against surgical intervention in border line cases.

Probably right heart catheterization should be done routinely, as an index to the severity of pulmonary hypertension. As the long term results of surgery are not yet known, surgery has not been advised unless there has been progression of symptoms on good medical management. As a

result, only late group 3 or group 4 patients have been accepted for repair.

OPERATIVE TECHNIQUE

The patient is placed in a supine position on the table with the right chest elevated approximately 30 degrees by a blanket roll extending from the thorax to the hips. A curved inframammary incision is then made and the right pleural space entered through the bed of the resected or stripped 5th rib. The sternum is divided transversely at this level, care being taken not to enter the left chest.

The pericardium is then opened parallel to and approximately 1 to 2 cm. above the phrenic nerve. A T-extension of this incision is then made toward the sternum and all edges reflected with stay sutures.

If there is any suspicion of aortic insufficiency in the preoperative work-up, the aorta is prepared for cross-clamping. Tapes are passed around the cavae and the right femoral artery is prepared for cannulation. The cavae are cannulated through separate purse-string sutures in the right atrium. Initially a third cannula was placed in the right atrium for drainage of coronary sinus blood, but this has been abandoned in the more recent cases as not necessary.

There is almost always sufficient left atrium presented in the operative field to facilitate easy access to the mitral valve. We have found it necessary to approach the valve through the right atrium and interatrial septum in only one case, and much prefer the direct left atrial approach.

The usual connections are made to the pump oxygenator and total cardiopulmonary bypass instituted.

Perfusion. The Kay-Cross rotating disc oxygenator has been used in all cases. A perfusion rate of 2400 cc. per square meter of body-surface area has been arbitrarily set as the ideal flow rate. In practice, however, a flow that will give a venous oxygen saturation in the range of 70 per cent has been followed. This will in some cases be less than the flow calculated on body-surface area. A mixture of 97½ per cent oxygen and 2½ per cent carbon dioxide is supplied to the oxygenator.

After finding large particles of calcium in the venous filter, and fine calcium sand in the arterial filter after repair of a heavily encrusted valve, we replaced the metal screens in the venous filter with a stainless steel scouring pad. This has eliminated fine calcium sand in the arterial filter, which we feel is a potential source of emboli. Carbon dioxide is also used in the operative field as one precaution against air emboli.

Repair. After the patient is on total bypass, a small incision is made directly into the left atrium and an exploring finger introduced to assess the valve. This initial examination is quite important in evaluating fully the degree of insufficiency, its location, the presence of associated stenosis and valve mobility before the atrium and left ventricle are fully decompressed. The incision is then extended, the cardiotomy suckers introduced and the valve visualized. The various factors present in the valve are re-evaluated and a plan of repair made.

In those valves which contain heavy encrustations of easily dislodged calcium, we have meticulously removed this calcium before disturbing the valve. If there is associated commissural fusion, this fusion is relieved by finger fracture, or by the use of a heavy instrument introduced between the valve cusps and spreading. Care is taken to open the commissures to the fullest extent. Any associated subvalvular stenosis should also be corrected at this time. It is surprising in some cases what this degree of mobilization of the valve will do to decrease the insufficiency present. If there are any large plaques of calcium present in the septal leaflet, these are also broken up, and any rolled edges of either leaflet are also mobilized.

Valuable time will be saved if, at this stage of the procedure, ruptured chordae are looked for. We have encountered one case of chordal rupture, and in this instance the ruputure was in the midportion of the posterior leaflet. The tissue at the site of rupture was attenuated and the insufficiency was easily corrected by rolling this tissue in with interrupted sutures, forming a baffle much as described by Guidry.[12] More recently, McGoon[13] has reported repair of flail septal leaflets by a plication procedure.

Annuloplasty. After complete mobilization of the valve, the annulus can be approximated, with no. 2 silk sutures. The annulus is not always readily identifiable, and care must be taken to be certain that the annulus is incorporated in the sutures, and that the sutures are not placed in the left atrial wall. The exact location of the

annulus can be better identified by inserting a finger under the valve leaflet, clearly identifying the junction of the leaflet with the atrial and ventricular wall. Several sutures can then be placed, usually at the posterior medial commissure, and snugged down. The degree of insufficiency remaining is then evaluated and if necessary, further sutures taken, care being taken not to reduce the valve orifice too much. We have felt that a residual orifice that will accomodate two fingers is adequate.

Baffles. In some cases, after adequate valve mobilization and annuloplasty, there will be more insufficiency remaining than one would desire. If this should be the case, a baffle of compressed Ivalon, or a roll of Teflon fabric can be sutured beneath the posterior leaflet at the site of insufficiency. In our hands, this has been somewhat difficult to accomplish technically, and has not in every instance given the desired result.

As the valve is made more competent, care should be taken to hold the valve incompetent periodically, to prevent the accumulation of air in the left ventricle. After the repair has been accomplished, a clamp is inserted through the valve and the left ventricle and atrium are allowed to fill. We have then aspirated the blood from the atrium, and revisualized the valve for a short period of time to fully assess the repair when the left ventricle is in a more distended condition. If the repair is then deemed satisfactory, a clamp is replaced into the ventricle, the ventricle, and then the atrium, is allowed to fill.

The atriotomy is then closed, beginning a running suture from either extremity of the incision, toward the center and the incision closed, except for a small portion in the midline. A Beck clamp is then used to close the incision, leaving a small portion unsutured until the patient is taken off of cardiopulmonary bypass. This is done in order that the left atrium may be decompressed if there is any tendency for the heart to dilate in the immediate post pump period.

Occasionally, immediately after opening the atrium, the volume of blood present will appear to be more than the cardiotomy suckers will handle. We have found that if the flow rate will be momentarily reduced, the atrium can be aspirated dry and kept so, after returning to the previous flow rate. If the atrium cannot be maintained adequately dry to allow good visualization of the valve, one should suspect aortic insufficiency. It may then be necessary to occlude the aorta intermittently. In these large precarious hearts even this momentary interruption in the heart's blood supply may lead to ventricular fibrillation and thus increase the problem of taking the patient off cardiopulmonary bypass.

RESULTS

To date, 8 patients with pure or predominant mitral insufficiency have been operated on. The first patient operated on (fig. 1) had pure insufficiency and annuloplasty at the posteromedial commissure alone was performed. The immediate postoperative result was gratifying and her symptoms markedly improved as evidenced by the decrease in cardiac size (fig. 2) evident 10 days postoperatively. This patient continued to show improvement for 6 months postoperatively, at which time she had a gradual reoccurrence of all preoperative signs and symptoms and again went into congestive heart failure. She continued to show deterioration in the hospital, despite medical management, and was reoperated.

At reoperation, it was found that one of the two previously placed sutures in the posterior medial annuloplasty had not been correctly placed, and there was a break down of the repair. The annulus was reapproximated and a baffle placed beneath the posterior leaflet. This improved the insufficiency but did not completely correct it. Bypass was slowly discontinued. The heart appeared distended, but its output seemed adequate. During chest closure there was progressive distention of the heart and finally ventricular fibrillation. Postmortem studies confirmed that the incompleteness of the second repair was the probable cause of her death.

A second operative death was felt to be directly related to associated aortic insufficiency. Preoperatively it was recognized that the patient had aortic insufficiency but this was not felt to be hemodynamically significant. However, immediately after going on complete cardiopulmonary bypass and opening the left atrium, a dry operative field could not be obtained in spite of cardiotomy suction of 3000 cc. per minute. The aorta was cross-clamped and the heart immediately fibrillated. Annuloplasty was then accomplished, but the effectiveness of the repair could not be fully evaluated in the fibrillating heart. Several attempts were made to take the patient off of cardiopulmonary bypass but the left ventricle

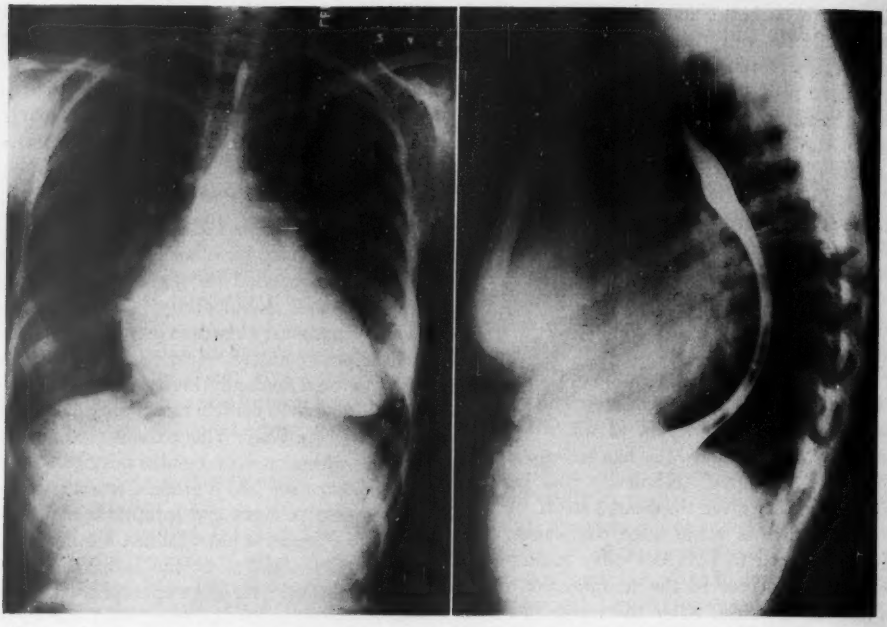

FIG. 1. Preoperative roentgenograms of 19-year-old white woman with pure mitral insufficiency.

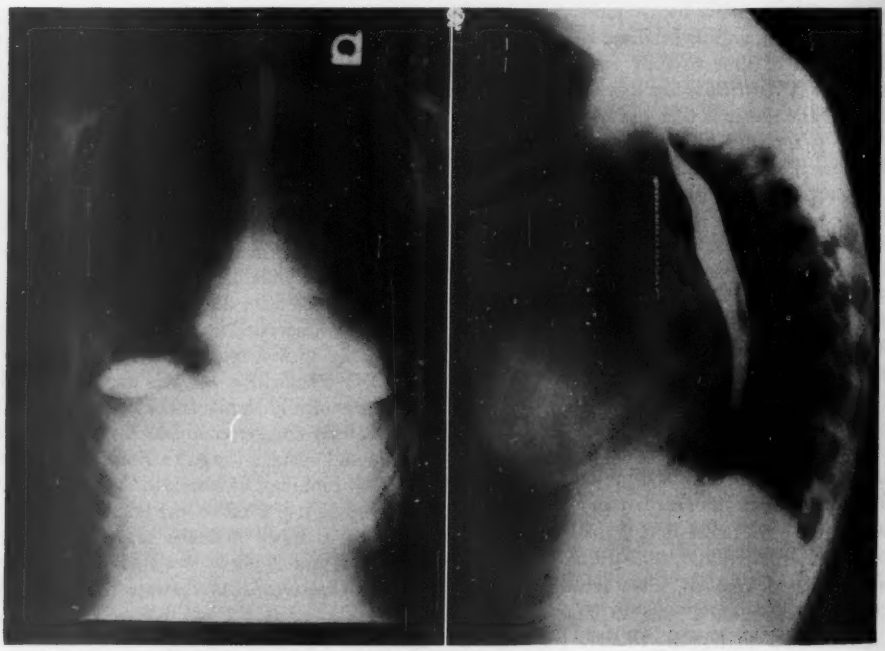

FIG. 2. Postoperative roentgenograms of the patient in figure 1

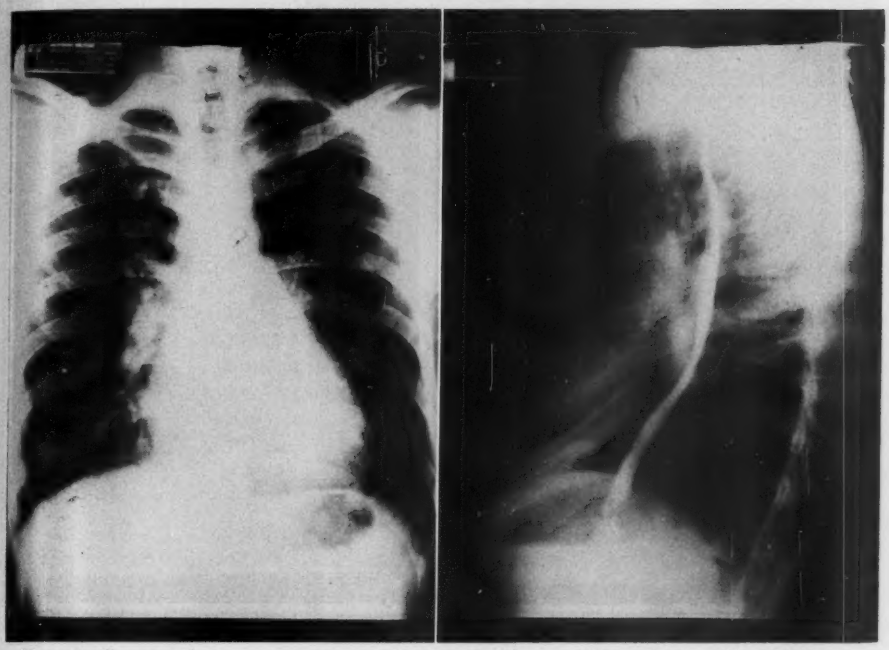

FIG. 3. Preoperative roentgenograms of 21-year-old man with predominant insufficiency

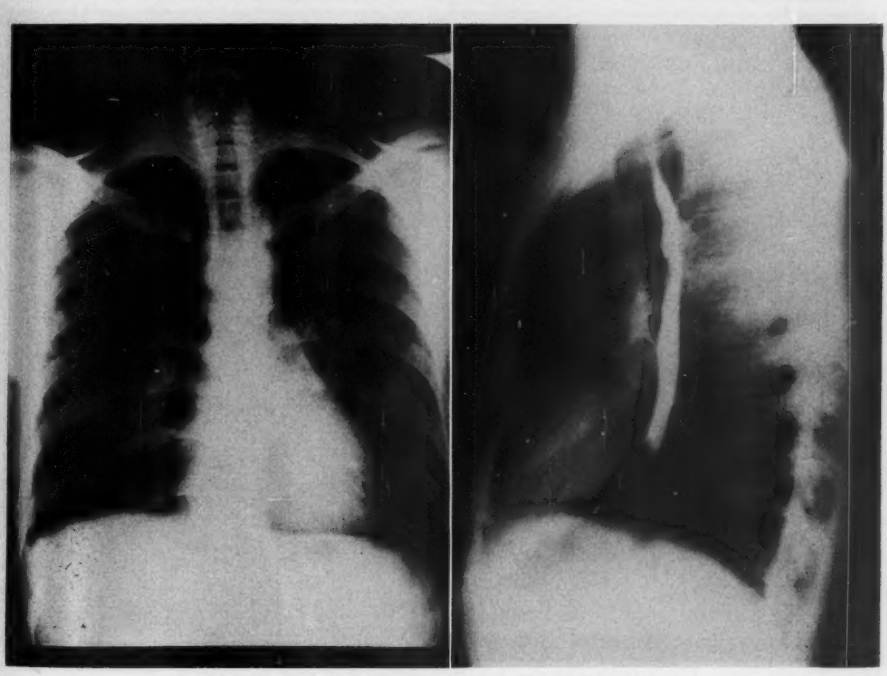

FIG. 4. Postoperative roentgenograms of patient in figure 3

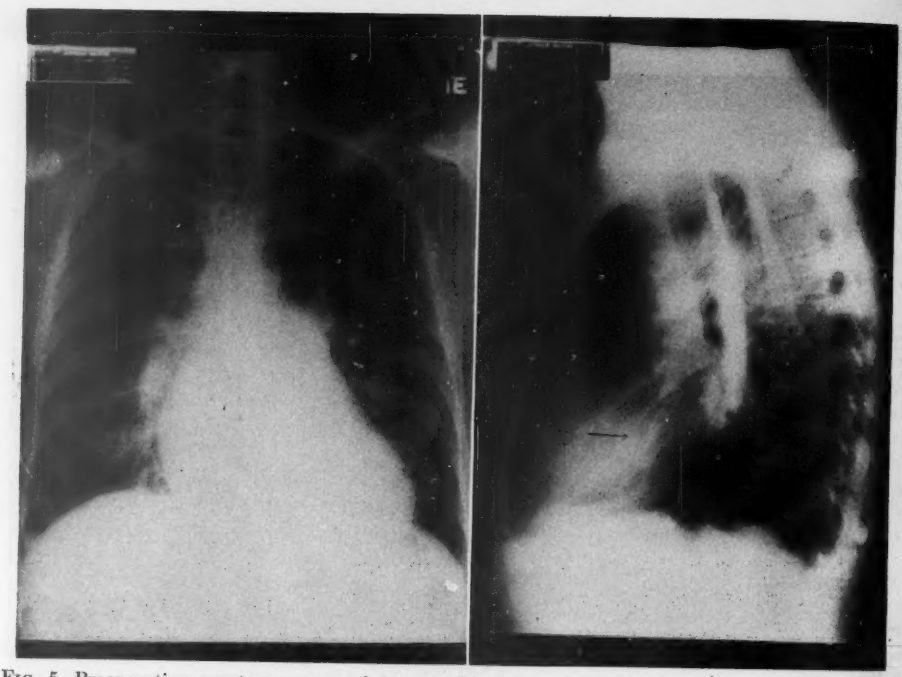

FIG. 5. Preoperative roentgenograms of 44-year-old white man with predominant mitral insufficiency. *Arrow* points to calcified mitral valve.

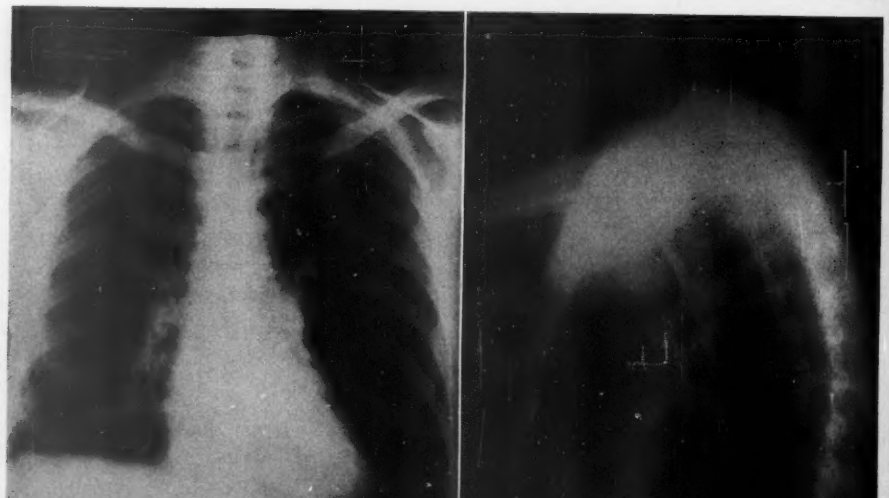

FIG. 6. Postoperative roentgenogram of patient in figure 5

distended with each attempt and finally became unresponsive.

A third patient expired on the sixth postoperative day, after successful repair of a ruptured chorda to the posterior leaflet. This death was felt to be directly related to postoperative acute tubular necrosis. At postmortem examination, the mitral valve was fully competent.

The other five patients are living and well, and all have shown improvement over their preoperative conditions (figs. 3 to 6). Three of these patients have been followed long enough to feel that their improvement is permanent, whereas two cases are yet too recent to fully evaluate. These two patients, however, have shown increase in exercise tolerance and an over-all sense of well being.

DISCUSSION

This small initial group of patients leads us to believe that the open approach to repair of mitral insufficiency is feasible and can be accomplished with an acceptable operative mortality when the serious nature of the patient's condition is considered. Certainly, some valves will be encountered which are so severely damaged as to be technically beyond repair. In other instances, a repair somewhat less than desired may have to be accepted. These instances will account for the operative mortality and poor results following surgery. As we have no way of predicting preoperatively which valves can be repaired, the procedure will necessarily carry a slightly higher mortality rate than desired, and for this reason, should most likely be reserved for those patients who have shown definite progression of symptoms.

Those patients encountered with associated aortic insufficiency will continue to pose a real technical problem even if the insufficiency is felt to be hemodynamically insignificant. As pointed out by Merendino[11] an aortic valve which is the site of hemodynamically insignificant insufficiency preoperatively, assumes the proportions of markedly significant insufficiency when placed on total cardiopulmonary bypass.

After our unfortunate experience with aortic insufficiency described above, in a second case in which there was evidence of insufficiency, preparations were made for direct coronary perfusion and myocardial hypothermia in the event that the insufficiency proved to be significant at the time of perfusion. A momentary cross-clamping of the aorta was all that was required to allow good visualization of the valve and, although there was obvious regurgitation while on the pump, it was not more than the two cardiotomy suckers could easily handle. In the future we feel that facilities for direct coronary perfusion should be available in all cases.

As these hearts are in a very precarious position one must be prepared to spend the time necessary to repair these valves in an unhurried fashion. This of course requires a pump oxygenator system that will allow perfusions for a long period of time. Our time on the pump has varied from 40 minutes to 2 hours. This brief experience also substantiates the fact that in order to insure survival the valve's function must be improved. If there is any delay in the heart taking over its function following bypass, one should immediately suspect that the valve repair is less than desired.

SUMMARY

1. A brief review of the pathologic anatomy of mitral insufficiency and the selection of patients for repair has been presented.

2. The surgical technique of repair and the results of operation are given.

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